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THE JOURNAL
of
The Maine Medical Association

VOLUME 57 JANUARY, 1966 NUMBER 1

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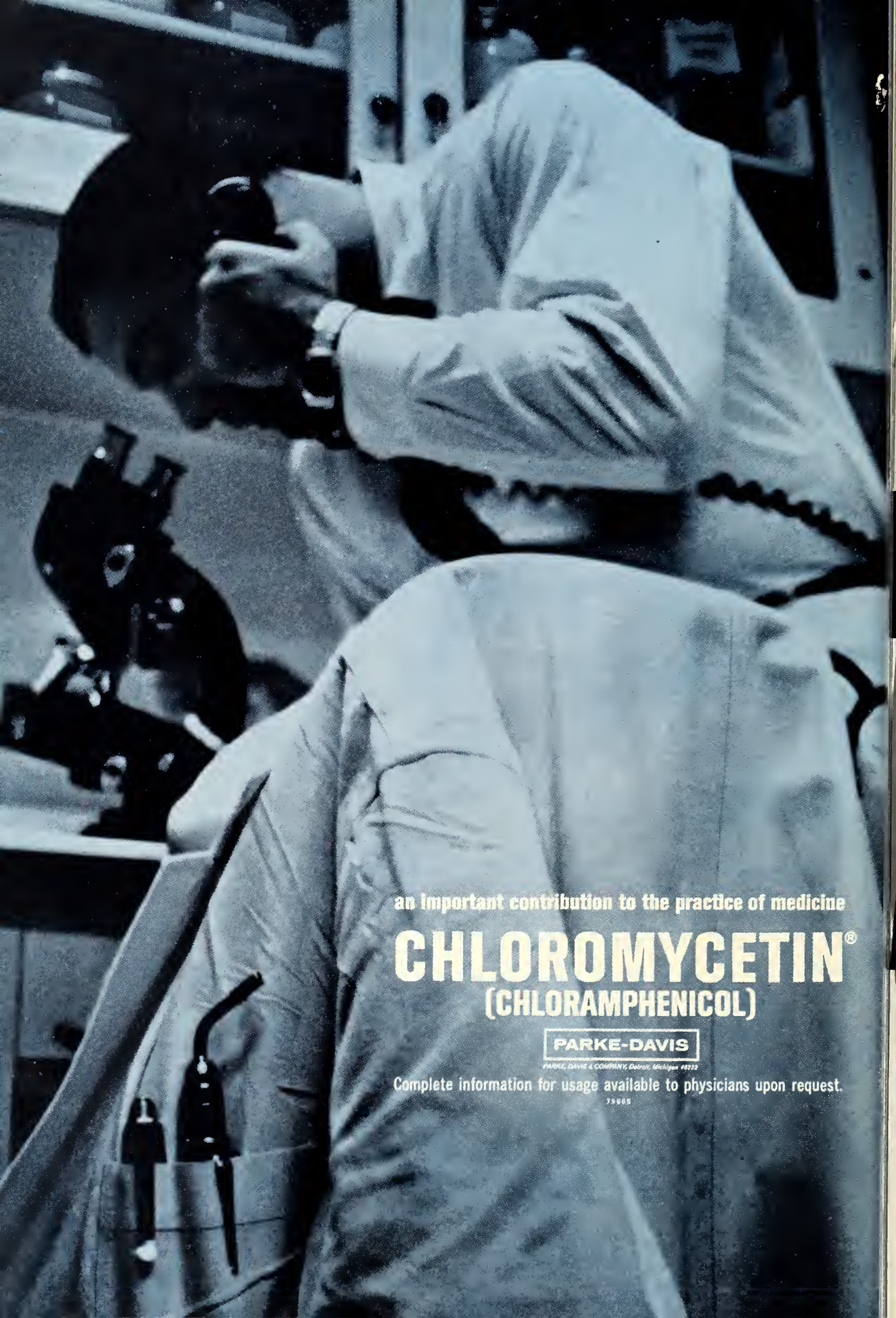
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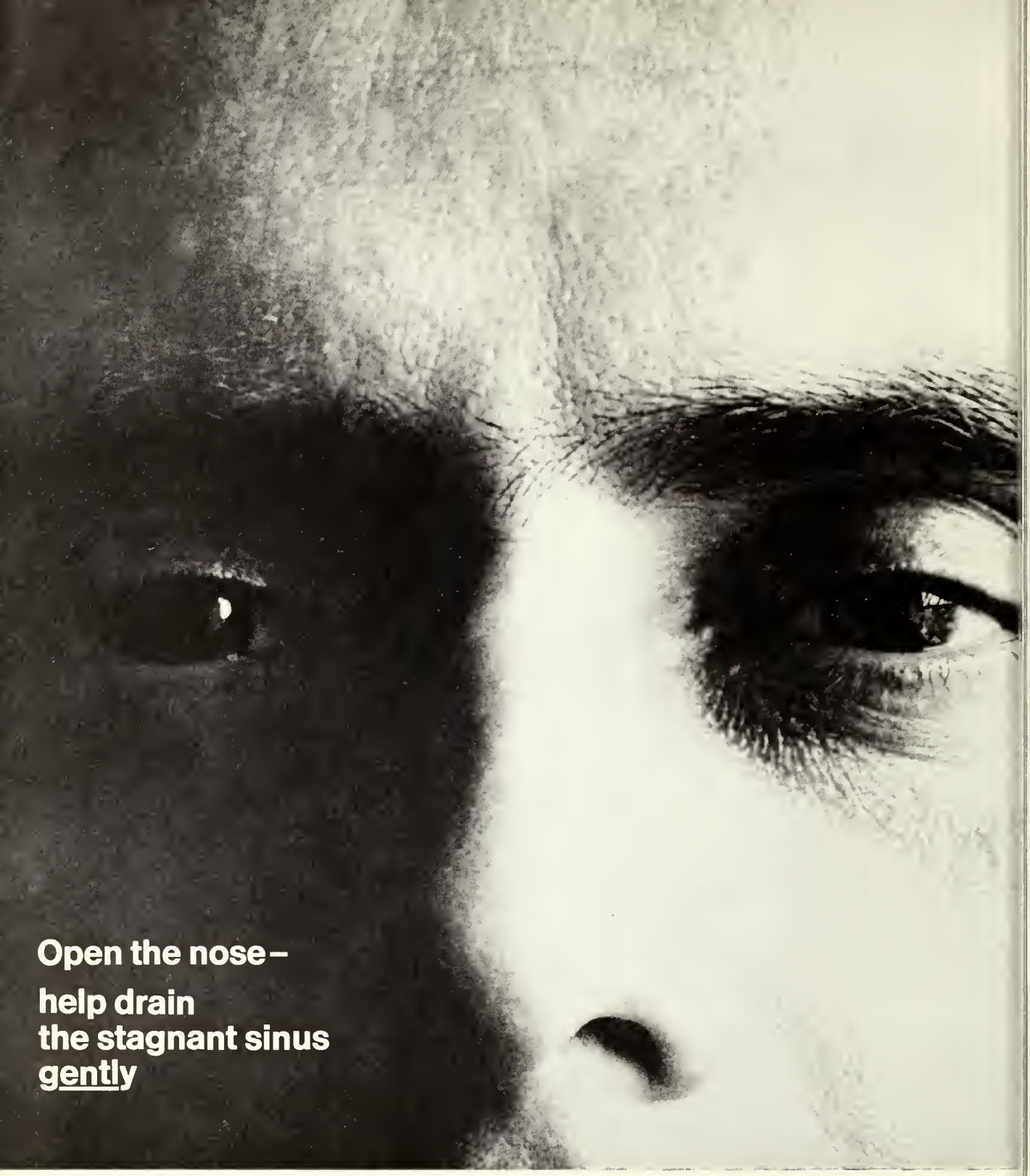
Literature on indications and dosage available on request.

(1) Frykman, H.M.: Minn. Med., Vol. 38, Jan. 1955. (2) Poth, E.J.: The J.A.M.A., Vol. 163, No. 15, April 13, 1957. (3) McGivney, J.: Texas State Jour. of Med., Vol. 51, No. 1, Jan. 1955. (4) Stern, F. H.: Jour. of The Amer. Ger. Soc., Vol. 11, No. 3, Mar. 1963. (5) Weekes, D. J.: N.Y. State Jour. of Med., Vol. 58, No. 16, Aug. 1958. (6) Abbott, P.L.: Jour. of Oral Surg., Anes. & Hosp. Dental Serv., Vol. 19, July 1961. (7) Weekes, D. J.: E.E.N.T. Digest, Vol. 25, No. 12, Dec. 1963.

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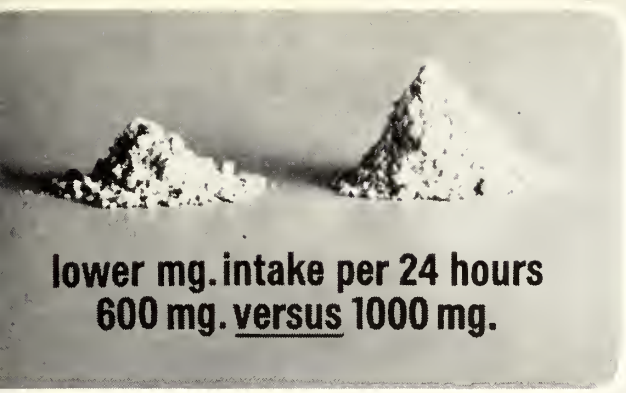
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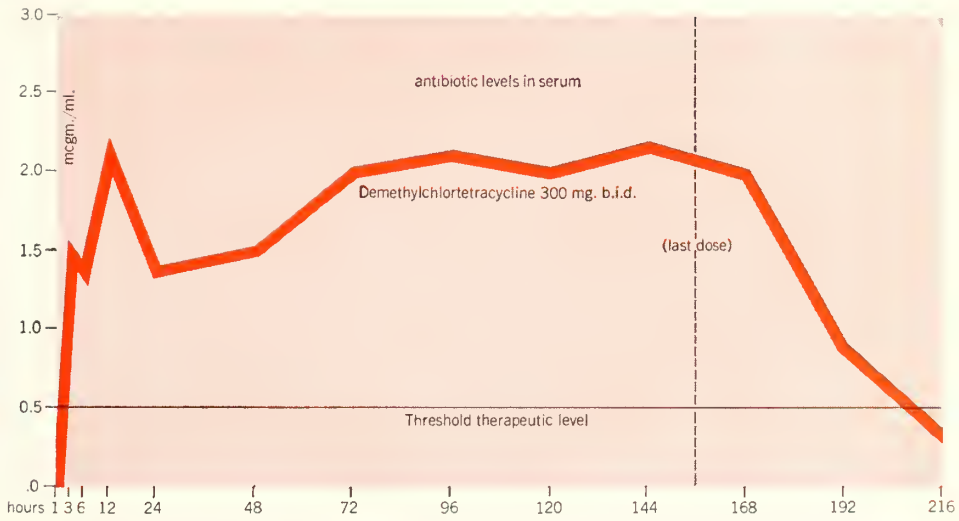


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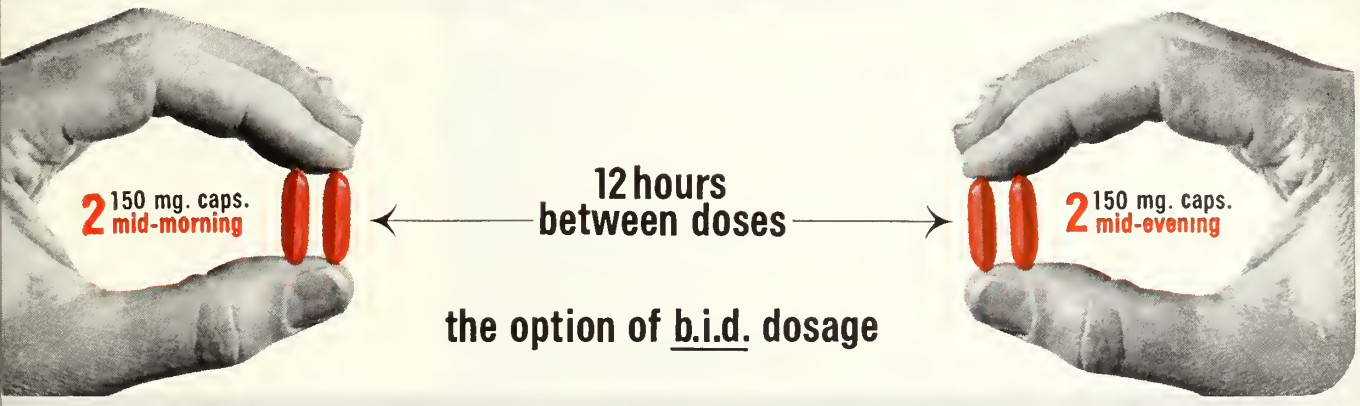


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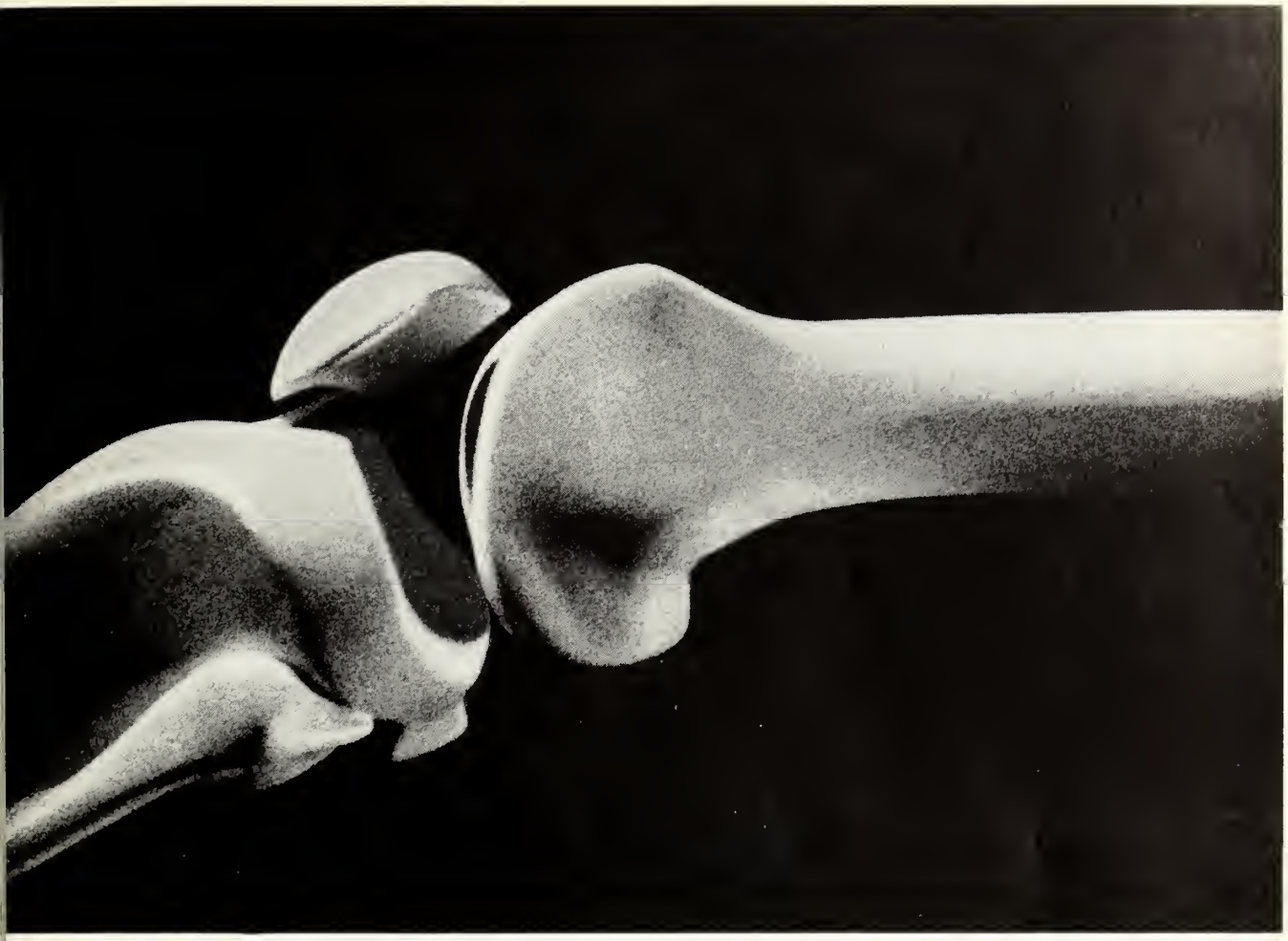
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recautions
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President's Address*

Thomas A. Martin, M.D.**

Fellow physicians, guests and friends: As is customary, and also mandatory, according to the by-laws of your Association, one of the duties of the president is to address you at the Annual Convention. Begging your indulgence, I will attempt to be as brief as possible:

Medicine, with its many disciplines, is a very complex affair, and with the ever-mounting advances in newer techniques, both diagnostic and therapeutic, is becoming more so.

As a result, the American people are the recipients of the best medical care in the world, and yet, what is our image in the public mind? I am sorry to say, my friends, that at least on the national level, it leaves much to be desired. However, on the local level, and in our everyday practice, this is not true. Hence, I believe it is time for us as a profession to do some soul-searching, and ask ourselves a few questions:

> Where are we failing in our rapport with the large majority of the public?–Or, are we failing in this rapport? Are we falling down in our communications, despite our educational program? Have we become so engrossed in the scientific aspects of our profession that we have lost the art of medicine, which our predecessors professed to such a high degree?

Frankly, I can see no conflict between the two since the art of medicine is simply the natural, human, charitable concern of one person for another when he is sick and troubled. Personally, I do not believe this for a second; on the contrary, I feel that the physician professes this to the "nth degree."

But, as all doctors know, we have been trained to be objective, which is so necessary in our work, and therefore, many times we give the wrong impression.

There is not a doctor sitting out there in this audience who has not gone home with a heavy heart and a troubled mind when failure, instead of success has crowned his best efforts.

If we have established that the physician has not lost his art, nor his humanity, what are some of the factors which are contributing to the loss of the personal relationship between the patient and the doctor? I will try to enumerate these causes as I see them:

> First, as a result of the great progress made in the scientific and technical aspects of medicine, many procedures and tests must of necessity be performed in the hospital, which of course, brings it further and further into the practice of medicine.
>
> Secondly, many of the above-mentioned procedures involve team-work between the doctor and para-medical personnel employed by the hospital.
>
> Thirdly, with the advent of complicated tools, which the physician uses, hospital costs have, of necessity, spiralled, and the third party, or vendors of services, have entered the health field, such as the insurances carriers, governmental agencies and the like.
>
> Finally, at least in our larger urban hospitals, the Emergency Divisions have assumed many of the prerogatives and duties of the general practitioner or family physician.

In our experience at the Maine Medical Center in Portland, it has been found that actual emergencies are in the neighborhood of 40%, and that general medicine accounts for the remaining 60% of the case load. This situation has not developed because the hospitals have deliberately attempted to corral these patients, but have simply been fulfilling their obligations and duties to the communities.

From the foregoing observations, and doubtless there are many more which I have not considered, the patient-

*Presented at the 1965 annual session of the Maine Medical Association.

**President, Maine Medical Association, 1964-1965.

physician relationship has become more impersonal. This, I believe, has occurred because of the ever-increasing case loads and demands, and not because the physician necessarily desires it.

Therefore, my friends, I feel we are going to hear and read more about medicine's image, and I am going to venture the prediction that this image will become more tarnished and less flattering in the future.

We can allow ourselves to become resentful, bitter and disillusioned; maybe even with some justification or satisfaction of sorts. But, will such action make our image less tarnished and more flattering? My answer to this is a firm NO.

Ours is a proud heritage, a noble calling and an honorable profession. We have an obligation to the many dedicated physicians who have preceded us, and a duty to those who follow, to maintain the principles and high standards of our profession, with malice towards none and charity for all.

Therefore, regardless of the cost, let us resolve to remain true to the noble calling and high traditions of the profession, of which we are privileged to be members.

Studies of Uremia*

Stanley G. Dienst, M.D. and Betty Morris

Uremia or the retention of urinary solutes in the blood is a commonplace syndrome associated with renal insufficiency. It is characterized by increasing weakness, muscular twitching, slurred speech, mental clouding, and coma. The progression of these signs generally parallels the elevation of blood urea concentration. In the later stages hyperkalemia and phosphatemia with increasing metabolic acidosis accompany rapid clinical deterioration.[1] This study is an analysis of the plasma organic acids in patients with advanced uremia. The findings are discussed together with preliminary results of current work on uremic animals. These studies are undertaken to determine the nature of intracellular changes which occur in the uremic patient.

The organic acids are the metabolic intermediates common to the oxidation of carbohydrates, fats, and proteins (Fig. 1). Their total concentration in uremic plasma has been previously estimated by measurements of acid-base balance. Hyman has reported values ranging from eight to twenty-five meq/L.[2] The identification of the individual acids has been undertaken using paper chromatography,[3] colorimetric chemical reactions,[4] and other methods.[5] However, there has been no final concensus regarding the proportion of individual acids. This study uses chromatographic methods to partially separate the individual acids and to measure them quantitatively.

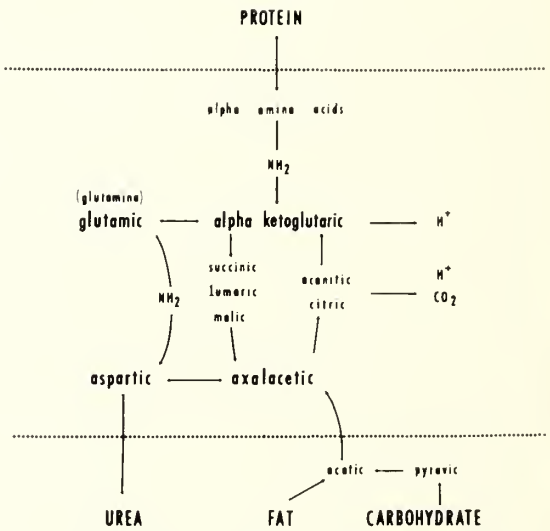


Fig. 1. Intracellular Relationships of Organic Acids. This is an outline of the relationships of organic intermediates common to fat, carbohydrate, and protein metabolism. The final oxidation of these compounds is dependent on the transfer of hydrogen ion to molecular oxygen. This transfer system is localized in the wall of mitochondria (see text).

Methods

Organic acids are concentrated by evaporating the neutralized protein free filtrate of plasma (protein precipitated by sodium tungstate and (H^+) ion exchange resins). This sample is acidified and absorbed into silica gel. The individual organic acids are eluted from the gel sequentially with an increasing concentration of butyl alcohol in chloroform according to the procedure first applied to tricarboxyllic acids by Bulen, Varner, and Burrell.[6] The organic acids are recovered and titrated in one hundred and fifty 3.0 ml. fractions (Table 1). In addition to identifying the plasma acids by the fraction numbers in which they are eluted from the silica gel column, they are also checked against standard acids by paper chromatography (Table 2). Varying quantities of formic, pyruvic, α-ketoglutaric, and β-hydroxybutyric acids are not recovered by this method.

*From the Metabolic Section, Cardiovascular and Renal Research Laboratory, Maine Medical Center, Portland, Maine.
This investigation was supported by Grant No. AMO 6095 PET from the National Institutes of Health.

TABLE 1

Percentage Recovery of Known Standard Acids Through Entire Procedure

Acid	*Fraction Numbers*	*% Recovered from aqueous electrolyte solution*	*Percentage recovered when added to plasma (Above initial values)*
Acetic	10- 18	97 (X4) *	98 (X1) *
Formic	23- 32	77 (X5)	75 (X1)
Fumaric	19- 30	95 (X2)	85 (X1)
Glutaric	20- 30	101 (X2)	—
Lactic	34- 43	123 (X3)	103
Succinic	36- 48	113 (X4)	—
α-keto	37- 50	76 (X3)	—
Aconitic	46- 55	87 (X3)	96
	64- 77		
Malic	83-103	101 (X4)	93
Citric	102-134	98 (X3)	100 (X2)

*Number of Determinations

TABLE 2

A Comparison of Plasma Unknown and Standard Acid Rf's by Paper Chromatography

Recovery from Plasma by Column Chromatography		*Paper Chromatography*	
Fraction Number	*Known Standard Acids*	*Rf standard acids*	*Rf Plasma*
15- 22	Acetic	.47 DEAE	.47 DEAE
28- 36	Formic	.50 DEAE	.49 DEAE
28- 36	Fumaric	.80	.80
39- 52	Lactic	.72	.72
39- 52	Succinic	.78 thin layer	.77 thin layer
45- 49 trans	Aconitic	.21	.22
64- 78 cis		.73	.74
78-101	Malic	.40	indefinite
102-125	Citric	.29	.29

Results

As shown in Table 3 the total organic acids in plasma from ten normal subjects fell in a narrow range from 3.4 to 7.0 meq/L. Values from the uremic patients varied from 7.5 to 21.5 meq/L. The first eight blood samples showed metabolic acidosis by pH and CO_2 measurements and are the only ones used to make up the average values for organic acids in uremia. Lactic, *trans* aconitic, malic and citric acids were the predominant acids in the uremic group. A portion of the aconitic acid represents isocitric present before acidifying the plasma with exchange resins.

All patients were bedfast with varying degrees of mental confusion, slurred speech and muscular twitching. Those with the most pronounced nervous system manifestations generally had the highest total organic acid levels. There was no consistent correlation of the acid levels with either BUN elevation or severity of oliguria.

Discussion

The elevation of organic acids in the plasma of uremic patients[9] immediately suggests an alteration in their steady state concentration caused by an inhibition of their further oxidation. As indicated in Fig. 1 the pertinent oxidative enzymes are concentrated in the mitochondria. It is mainly at this site that specific dehydrogenases and decarboxylases progressively oxidize the organic acids through the tricarboxyllic acid cycle. The hydrogen ions from the acids then reduce coenzymes and flavoproteins in the mitochondrial wall. The elec-

TABLE 3

Summary of Organic Acids in Ten Normal Subjects and Ten Uremic Patients

							Total meq/L	*Organic acids* *Acetic*	*Formic (fumaric glutaric)*	*Lactic (Succinic α keto)*	*Aconitic (cis-trans)*	*Citric (Malic)*
Ten Normal Subjects					Average		5.2	0.4	1.0	1.5	0.9	1.4
					Normal Ranges		3.4-7.0	0.1-0.9	0.5-1.5	0.8-2.3	0.5-1.4	0.9-1.9
Ten Uremic Patients					Average		10.3	0.7	1.3	3.4	2.2	2.7
Patient		Pathology	BUN (mgms %)	pH	CO_2	pCO_2						
R.T.	43	Papillary Necrosis	196	7.31	9.	17.	7.5	0.6	0.3	1.9	2.2	2.5
F.T.	66	Pyelonephritis	176	7.46	25.	35.	8.0	0.3	1.2	4.1	1.3	1.1
A.N.	50	Tubular Necrosis	149	—	5.	—	11.2	0.9	1.2	3.5	2.7	2.9
M.C.	44	Papillary Necrosis	127	7.34	14.	25.	8.2	0.6	2.2	2.4	1.7	1.3
A.H.	60	Tubular Necrosis	185	7.46	16.	22.	18.9	1.0	1.8	9.1	2.2	4.8
G.E.	37	Pyelonephritis	170	7.35	8.	14.	7.7	0.5	0.9	2.0	1.4	2.9
G.E.	37	Same Patient	142	7.30	19.	37.	13.1	1.2	1.5	3.2	3.2	4.0
W.H.	26	Pyelonephritis	189	7.34	15.	27.	7.0	0.4	0.1	2.4	2.1	2.0
I.T.	55	Pyelonephritis	168	7.37	17.0	30.	11.5	0.9	2.2	2.3	2.9	3.2
W.H.	26	Same Patient	216	7.14	16.	43.	7.6	1.0	1.1	2.2	2.1	1.2
P.M.	43	Hypertensive Nephrosclerosis	209	6.91	10.	46.	21.8	3.6	3.4	9.2	3.9	1.7
P.M.	43	Same Patient	178	7.16	13.	36.	21.2	2.3	1.8	11.0	3.0	3.1
B.S.	68	Tubular Necrosis	150	7.18	17.	43.	9.6	0.7	0.9	2.8	2.9	2.3

trons released in the reoxidation of flavoprotein are transferred through a fixed sequence of cytochromes in the inner mitochondrial wall to oxygen. Though many factors influence the rate of hydrogen and electron transfer it is normally sufficient to prevent the accumulation of acids.

To evaluate the efficiency of this transfer process in uremia, we have used mitochondria isolated from uremic rat liver tissue. Urea concentrations have been kept constant (250-300 mg% urea nitrogen) from the nephrectomized animals original blood level to final mitochondrial suspensions. Fluorometric techniques have been used to follow the rates of reduction and oxidation of intramitochondrial coenzymes[7] using different organic acids as substrate.

Mitochondria isolated from liver tissue of normal rats and oxidizing different organic acid substrates maintain a ratio of reduced to oxidized coenzymes $\left(\frac{NADH}{NAD}\right)$ * of about 1.0. The addition of phosphate acceptor as adenosine diphosphopyridine nucleotide stimulates hydrogen transfer and causes a transient oxidation of NADH to NAD.[7] Mitochondria from uremic animals (in a media containing urea) show a progressive oxidation of all the coenzyme and a rapidly diminishing response to phosphate acceptor. Less striking but similar results are obtained by exposing normal mitochondria to urea (300 mg%) for four to five hours.

In studies of oxygen uptake it is apparent that liver mitochondria from uremic animals consume oxygen more rapidly than controls. The increase is greatest in the presence of phosphate acceptor. This suggests that more oxygen may be required for phosphorylation by uremic mitochondria. Further work on this aspect is in progress.

To date these observations do not form a complete pattern. They certainly do not support the thesis of an intramitochondrial block to electron transfer (from coenzyme I and flavoprotein to oxygen). Actually they indicate an increased rate of transfer. This leaves the original questions of why tricarboxyllic acids are elevated in uremic patients unanswered. It must be continuously kept in mind that findings in isolated suspensions do not necessarily represent the reactions of mitochondria in the cell or in the patient. However, they do indicate that the mitochondria undergo definite changes as the result of the uremic process.

Finally a word about lactic acid. The increased values of lactic acid in uremic patients may be explained as a secondary effect occurring extramitochondrially.

Normally the level of lactate is proportional to the ratio of reduced to oxidized extra-mitochondrial coenzyme I $\left(\frac{NADH}{NAD}\right)$.

Pyruvate + NADH ($\uparrow$) $\longleftrightarrow$ Lactate ($\uparrow$) + NAD

In the uremic the excess acids (malic and isocitric indirectly), can increase the proportion of reduced coenzyme (NADH). This in turn reduces pyruvate (produced from glycolysis) to give an elevated lactic acid level. In this respect advanced uremia may be similar to other advanced metabolically decompensated states where changes in intramitochondrial equilibria are finally manifest as acidosis.

*NAD – Nicotinamide adenine dinucleotide.

Summary

1. Results of studies of organic acids in uremic patients are presented.
2. The rationale for further studies of mitochondria from uremic and normal animals is discussed. The preliminary results of these studies are outlined.
3. A note on lactic acid elevation as a probable consequence of tricarboxyllic acid elevation is included.

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Psychiatry and Criminal Responsibility*

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Society's awareness that a crime can be the result of mental illness or of defective intelligence, as acknowledged and reflected in our criminal laws, is relatively new.

In 1870, the State of Maine adopted the "right and wrong test" which was based on the well-known M'Naghten Rule (England-1843) ". . . To establish a defence on the ground of insanity, it must be clearly proved that at the time of the committing of the act, the party accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or, if he did know it, that he did not know that he was doing what was wrong."

In 1961, the Legislature of the State of Maine enacted a new test for establishing mental responsibility for criminal conduct. This statute, known as the Durham Rule, provides that an accused is not criminally responsible if his unlawful act was *the product of a mental disease or mental defect.*

This broad and generalized reference to mental illness or mental defect has a number of psychiatric presuppositions which I wish to discuss:

1. *Mental Illness*: The law implies that mental illness is a well-defined and generally accepted concept; that it has clear-cut lines of demarcation toward mental health; that it is a disease process which can either improve or deteriorate; that it has a demonstrable pathologic substrate.

2. *Mental Defect*: The law presupposes that mental defect is a clear-cut entity; that it has been defined acceptably; that it can be measured accurately. When one tests the reality of these presuppositions with respect to mental illness, one has to make some disconcerting observations:

The Diagnostic and Statistical Manual, Mental Disorders American Psychiatric Association 1952, on Page 1, refers to "diseases of the psychobiologic unit" after which specific conditions, presumably diseases, are listed as "disorders". Thus "disease" and "disorder" are used interchangeably.

Webster's dictionary defines "disease" as: A condition in which bodily health is impaired; sickness; illness; also, a malady; an ailment. The same source defines "disorder" as: 1. Lack of order; confusion; disarray. 2. Neglect of order or system; irregularity. 3. Breach of public order; tumult. 4. Sickness; ailment. v.t. 1. To disarrange; to confuse. 2. To disturb the natural functions of (body or mind); to derange.

I doubt that there is agreement between psychiatrists or even physicians as to what constitutes a "disorder" and what is a "disease", although this is conceivable. I do not think, however, that any physician will use "disease" and "disorder" interchangeably, implying a synonymous definition.

Since the psychiatrist will find himself in the legal halls of society, when involved in criminal procedures it is interesting to find out how the law defines "disease" and "disorder".

Black's Law Dictionary, 4th Ed., 1951, defines "disease" as: deviation from the healthy or normal condition of any of the functions or tissues of the body; an alteration in the state of the body or of some of its organs interrupting or disturbing the performance of the vital functions and causing or threatening pain and sickness; illness; sickness; disorder; malady; bodily infirmity. The same source defines "disorder" as: turbulent or riotous behavior; immoral or indecent conduct; the breach of the public decorum and morality. 2. Is usually a slight, partial, and temporary physical ailment.

Turning back to the Statistical Manual of the American Psychiatric Association, one notes that a group of disorders is classified according to symptoms with attempted etiological identification under "acute brain disorders" and "chronic brain disorders", both having demonstrable organic pathology.

The psychotic disorders come under the heading of "disorders of psychogenic origin or without clearly defined cause or structural change in the brain". They include manic-depressive, schizophrenic, and paranoid reactions. Under the same heading, we note the psychosomatic, psychoneurotic, and the personality disorders.

Thus, a major psychosis such as schizophrenia, a psychoneurotic anxiety reaction, a psychosomatic skin reaction, and a sociopathic personality disturbance, are all considered "disorders of psychogenic origin or without clearly defined cause or structural change in the brain". Does this imply that the patient with psoriasis the patient with an acute or chronic anxiety or phobic reaction, is mentally ill and should be excused if found guilty of a criminal offense?

Does this imply that the anti-social actions of the criminal are a product of mental illness?

The intent of the law surely does not favor such approach. In its present wording, however, it would leave the door wide open to an abuse of provisions that were meant to protect certain mentally deranged people against inappropriate punishment. Therefore, it is suggested that an Interdisciplinary Committee, composed of members of the Maine Medical Association and the Maine Bar Association, study the possibility of replacing the words "mental illness" by specific identifiable

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pathologic disease entities which should be included in the law.

In reference to "mental defect" the confusion is equally bewildering. The Statistical Manual of the American Psychiatric Association refers to this item as including "disorders due to unknown or uncertain cause with the functional reaction alone manifest; hereditary and familial diseases of this nature." (Note, again, the interchangeable use of "disorder" and "disease").

Sub-classifications into degrees of "mental defect" are offered and to be arrived at by testing. However, the test or tests to be used are not identified. This section reflects a degree of ignorance on the part of the profession that is not in keeping with our actual knowledge in 1965.

The Group for the Advance of Psychiatry in its Report #43*, in 1959, pointed out that the terms "mental retardation", "mental deficiency", "feeble-minded", "mental inadequacy", and "sub-normality", refer to a broad phenomenon indicating unusually slow or arrested development and decided to use the term "mental retardation", a national trend not yet reflected in the American Psychiatric Association Manual.

The Group for the Advancement of Psychiatry further acknowledges that "modern advances in the medical sciences have made us increasingly aware of the multiplicity of the factors in the etiology of mental retardation" and submits that known etiological factors "will fall into one of the following categories: somatic, cultural, social, psychological".

At any rate, it is our experience that some individuals with impaired learning ability, functioning in the borderline range of intelligence, or in the mild range of retardation actually have concurrent personality trait disturbances that are unrelated to intellectual functioning. If convicted of criminal offenses, these individuals are properly placed in correctional institutions regardless of the fact that their intelligence quotient may measure only 65 or 70. The rare occasion in this category is the patient who simultaneously suffers from an epileptic syndrome or a metabolic disturbance which probably should be covered under the section of specific diseases, replacing the words "mental illness", as proposed.

Patients with a mental retardation syndrome, whose disability places them in the trainable or dependent range of intellectual deficit are usually considered incapable of premeditated crimes and if found guilty of such a crime, it was most likely either the result of an accident or it was instigated and supervised by a competent person. In these cases, it is rare if, indeed, ever necessary, to place such a person under security provisions. Adequate social supervision as offered in institutional or group living as offered in nursing homes, sheltered workshops, etc., will suffice.

For didactic purposes I would like to summarize now that we have no valid or commonly accepted definitions of either "mental illness" or of "mental defect" yet these 2 terms are part of a law that presupposes that such definitions do exist.

Next, I wish to explore criteria used by psychiatrists when testifying as expert witnesses. The testimony given, at least in the majority of the cases, is based upon examinations that took place after the crime was committed. However, the psychiatrist is asked whether the respondent's crime, if, indeed, he committed one, was the result of "mental illness" or "mental defect".

Can a qualified psychiatrist, beyond any reasonable doubt, testify *in every case* to a condition of which he had no knowledge at the time of the alleged crime? Surely, there are many cases where this can be done. However, there are others where this may be difficult or even impossible.

In other words, the psychiatrist may be asked to testify to a "mental illness" that he did not witness at the time it was supposed to have existed and he may further testify that the crime committed was *"the result* of mental illness" even though the court has not even ruled that the crime was committed by the respondent whose "mental illness" the psychiatrist may be asked to testify has caused the crime. I wonder whether psychiatrists have this capability. I rather think not.

Schumacher†, and others, have recommended that the criminal procedure that leads to the establishment of guilt (or innocence) should precede a hearing on the absence or presence of illnesses which would cause the respondent to commit the crime of which he has been convicted. In this manner, the presence of guilt would be proven first and replace the current plea of "not guilty" but if guilty, "I am not guilty because I am mentally ill".

If a respondent has been found guilty of a crime by due process of law and thereafter pleads innocent by virtue of mental illness, the court should then refer this problem to a court-appointed review board of 3 qualified psychiatrists who should have the right and the resources to utilize the services of a qualified psychiatric social worker and a qualified clinical psychologist to assist in fact-finding and testing. The board should further be allowed to engage other specialists such as a clinical neurologist.

The diagnosis of psychiatric disorders is sometimes extremely complex and difficult and may require collaboration of several specialists. In this way we could ensure that every respondent has a comprehensive and sound evaluation regardless of what his financial resources are and regardless of whether his defense counsel has superior skill and ability or not, whether he has been retained or has been appointed by the court. This procedure, in my judgment, would constitute a vast improvement over present practices.

*Basic Considerations in Mental Retardation: A Preliminary Report, December 1959, New York.

†William E. Schumacher, M.D., Director, Bureau of Mental Health, Augusta, Maine, Personal Communication.

It would be incorrect, however, to anticipate that this would solve all problems. It will be a rare occasion, but distinct possibility, particularly when dealing with the so-called borderline case, when the professional board cannot agree amongst themselves. However, even then one could still refer the dispute to a Board of Appeals and always reserve the final decision to be made by the presiding judge.

Another safeguard for the protection of the mentally ill respondent should be added to the law:

> Presently, the convicted respondent's sentence is from "indefinite" to "life". His release, as stated above, is conditioned upon a statement signed by the Hospital Superintendent of "satisfactory proof that his discharge, or release in the custody of a relative or friend, will not endanger the peace and safety of the public". This, then, requires that the Superintendent is clinically concerned and involved with individual patients, which is rarely the case, as can be well-understood when one considers the size of our State Hospitals.

To eliminate the possibility of unnecessary and prolonged incarceration, I would like to see an amendment to the law to include that a respondent, confined to a mental hospital by court order, must be reexamined once every year by a court-appointed psychiatric review board and that the board's findings must be referred to the court for its determination whether further confinement is necessary or desirable. This would eliminate the need for the Superintendent, a single individual, with vast administrative obligations and involvements, to take a stand in a matter that should be left to clinical judgment. In addition, it would relieve a Superintendent of the real and anticipated responsibilities and consequences of having to make predictions about the conduct of an individual in months and years to come–a divine task not available to psychiatrists.

A further amendment that I have to recommend would provide for a prompt hearing within 30 days after a petition has been received by the court from either the respondent, his family, or the hospital Superintendent.

On April 26, 1963, we had to receive a young man who had been indicted and in whose case the Durham Rule was invoked. He was sent to a State Hospital for observation and later transferred to Pineland Hospital and Training Center. After careful consideration, it was determined that this patient's mild degree of retardation did not necessitate his indefinite incarceration. A hearing was arranged on December 23, 1964, and the patient was transported over 125 miles by the Director of Psychiatric Social Service, with a medical report, signed by the Superintendent. The court preferred to have a psychiatrist personally present to testify, if necessary, and refused, on these grounds, to hear the case. The patient was returned to the hospital and we have requested a new hearing by contacting the court-appointed attorney, to no avail. Almost six months later, this man is still waiting for an opportunity to regain his freedom. There may be understandable reasons for this delay. The fact remains that this man has not had access to the court for many months. Against this, the law must offer protection.

In summary, I wish to state:

1. that psychiatry must offer specific diseases to define the terms "mental illness" and "mental defect".
2. that these specific diseases, in all likelihood, can be identified as having caused the crime.
3. that a respondent should be found guilty or innocent of a crime before the presence of mental illness or mental defect are even considered.
4. that the presence of mental illness or mental defect be determined by a court-appointed board of qualified psychiatrists and allied specialists.
5. that a person committed by a Superior Court to be confined at a mental hospital as a result of a criminal offense be reexamined once every year, within 30 days of the anniversary of his commitment. This examination should be carried out by a court-appointed board of qualified psychiatrists and their findings should be reported to the court for further disposition, if indicated.
6. that a court must schedule a hearing within 30 days after receiving a petition from a respondent committed in this frame of reference to a State Hospital, his family, or his lawyer, or the Superintendent of the hospital in which he is confined, to determine the propriety of further detention at a mental hospital.

I would urge you, in all sincerity, to refer these recommendations to the appropriate committees of the Maine Medical Association for their consideration.

Group Therapy - An Aid to the Alcoholic

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The problem of the continuing management of the chronic alcoholic becomes increasingly important as this condition is widely accepted as a medical syndrome which must inevitably be handled, in part, by the general practitioner.

In one of my previous papers I discussed the treatment of the acute phases of alcoholism in a general hospital setting. Subsequent experience, involving over 200 such patients in a local community hospital, has already served to substantiate my earlier published views on this subject.

When a patient is discharged from the hospital environment, rehabilitation becomes a major problem. Over many years AA and its affiliates have done commendable work in the rehabilitation of these patients, but regretfully a number of alcoholics do not find their answers in this approach. For these, experience would indicate that group psychotherapy with eight to twelve patients, given once weekly, and extending over one to one and one-half hours, is extremely beneficial. I would emphasize that this can be achieved even in the limited space available in the average physician's office.

The physician who undertakes to treat these patients cannot progress far in successful psychotherapy without some understanding of the character structure of the addictive drinker. In the early part of this presentation I shall try to describe some of the general characteristics of these patients. Subsequently, I shall discuss the correlation of these characteristics with group therapy, both in a positive and negative sense.

There is no doubt that there is a tremendous psychological component to the disease we call alcoholism. Adequate treatment of this component of the illness may often permanently help the patient, providing a careful screening and evaluation of his situation has been undertaken. It is now generally accepted that very few, if any, alcoholics exist who do not have a deep-rooted psychological disturbance, and it is not at all unreal to speculate that this may be the true basis for their abnormal drinking patterns. This is particularly pertinent in the early phases of the illness, before secondary true addictive elements enter into the clinical picture.

Over many years, one of the greatest controversies in the field of alcoholism has centered around the question of a common personality pattern in these patients. As experience and knowledge has developed, most scientific workers in the field have come to believe that common patterns do exist and that a general alcoholic personality profile can be established. Intensive research on this subject was reported by the King's County Hospital, Brooklyn, New York, in 1956, and was perhaps best summarized by the following statement: "The theory that there is no unitary personality type among alcoholics, so widely supported in the literature, is rejected. To be sure, 50 alcoholic patients studied included virtually every adult psychiatric entity. Psychoses, neuroses and character disorders are represented. But it remains entirely possible for a common dynamic mechanism to be embedded in quite diverse personality integrations."

It is not my intention to suggest that all alcoholics can be "pigeonholed" into a set of constant personality and behavior patterns, but certain characteristic trends do recur again and again in this group of patients. An attempt to reduce to a common denominator the behavioral, biological and social characteristics of all alcoholics would be fraught with pitfalls since it would require too great a generalization on the one hand and, on the other, invariably involve the practitioner's own personal feelings and conclusions. Nevertheless, restricted generalizations can be made, and these are essential if one is ever to understand the dynamics of the alcoholic personality and, in turn, utilize this knowledge in the therapy of the patient and assist the alcoholic to understand his problems and to some extent fight his own battles.

Intensive studies by many dedicated investigators, and supported by my own findings, indicate a composite picture of the alcoholic in which certain basic features may be highlighted to serve as a guide in understanding the individual patient. In the order of their widest distribution, and therefore their significance, these characteristics are as follows:

(1) Dependency
(2) Inadequacy and immaturity
(3) Unique defense mechanisms

Dependency (Pathological Dependency) — This extreme need is expressed by many alcoholics in innumerable ways. It has even been said that the ways an alcoholic reflects his dependency are as numerous as the flowers in the fields. In general, the most common expression of dependency appears to be a compelling want to be taken care of, to be shielded from responsibilities, or from uncomfortable and unpleasant situations, frustrations and tensions. His reaction to these is his retreat to alcohol.

Korman and Stubblefield stated that "dependency ranked foremost in significance within each subject and their interpretation revealed as primary the need of an authoritarian or parent figure to cling to, to be guided by, and from whom reassurance, support and approbation were to be sought." Another authority felt that approximately 90% of all male alcoholics were maternally dependent individuals with inadequate, immature personalities.

My own experience, both on a professional and also group therapeutic relationship, indicates that this type of dependency and immaturity exists not only in males but also in a great percentage of females alcoholics.

Inadequacy and Immaturity — These two psychogenic dependencies are reflected in the alcoholic's seeming incapacity to resolve either inner conflicts or to deal with the external realities of life. The majority of these patients suffer from extreme tension and low tolerance towards stress, either as a result of a genetically weak ego or a poorly developed ego structure. If he cannot solve his problems quickly, he is unable to cope with them. This creates an intolerable anxiety, and as patience is a virtue he rarely possesses, he turns to alcohol as an antistress compound to make his existence more tolerable. His past experience indicates that nothing else has ever relieved the stress for him and therefore he goes to extreme measures to protect his drinking patterns. He quite often believes that he will die if he does not preserve this escape mechanism. This feeling, and on occasion conviction, leads him to develop a multiplicity of defense mechanisms which because of the intensity and the skill with which they have been developed are a unique characteristic of the alcoholic personality.

Defense Mechanisms — These may be divided into three parts: (1) Withdrawal (development of a schizoid personality)

In his reluctance to come to grips with problems and people, the chronic drinker holds himself apart, using the bottle as a shield. This leads to emotional estrangement and to the great loneliness which the alcoholic so often mentions. Interpersonal relationships are avoided, for they connote ties of fealty and responsibility which can only interfere with or disrupt his prime allegiance. The alcoholic anticipates any threat, real or fancied, to his alcoholic requirements and looks at every situation with suspicion and hostility. It is entirely possible that some of these paranoid manifestations arise from clumsy attempts by friends and relatives to disrupt his drinking habits.

(2) Dissimulation

The feeling that the alcoholic develops during his pathologic years of curtailment of his way of life from the outside causes him to use dissimulation. This technique of diversion of action has been honed to protective keenness and requires considerable insight and experience in the therapist for detection. Recognition of the presence of these devices should be constantly in the mind of the therapist and all statements made by the patient should be checked and cross-checked with the employer, immediate family and relatives, if at all possible. A favorite tactic resorted to by many alcoholics is to create crisis situations involving either the family or the law. These tend to compound and confuse the therapeutic milieu. Indeed, it may be truly stated "that every day becomes a new crisis in the life of an alcoholic." The alcoholic frequently tends to mislead the therapist into believing that drinking really presents no problems and that the whole situation is on the mend. To the astute listener, a failure of such devices will be revealed in an attempt to divert attention from the basic problem under consideration. The alcoholic will often attempt to bolster his statements with a discussion on an outside stress situation or individual which is causing him to drink excessively.

Clues to other defensive measures may be garnered in the forms of half truths truth distortion, truth omission and misrepresentation. Lying becomes habitual and eventually unrealistic and may lead to such confusion between reality and fantasy that the patient does not even recognize the truth, because of the belief he develops in the lie itself. The alcoholic's history may be very convincing and can divert and, indeed, deceive the therapist as to the real facts of the situation.

(3) Social Factors

A further factor of importance in treating the alcoholic in group therapy is the social element to his disease. The use of alcohol as a beverage and temporary escape from stress has been accepted for many centuries, and for the majority of people has never presented any real problem. This, in turn, has led to the acceptance in our culture of alcohol in various forms. We must recognize that without the availability of alcohol there could be no alcoholism. This fact was perhaps recognized during the prohibition era but this particular decade in our history clearly demonstrated that this was no solution to the alcoholic's problem. Since that time, the increasing stresses of our modern way of life have added to his problem, amplifying, not infrequently, psychological maladjustments that are absent in a more serene environment. Pressures from business relationships, especially in the executive and sales world, contribute to the development of alcoholism. This is perhaps exaggerated by the human need to conform and to be part of a group, so that drinking is accepted almost as a must if they are to be recognized in their own strata of society. It is not hard to see that this leads to the first step on the road to becoming an alcoholic.

The Alcoholic in Group Therapy — Group therapy for the alcoholic may either involve the private practicing physician or the institutional group therapy situation. Experience has shown that in private practice problems in group therapy arise which do not confront the institutional therapist. These special problems range from patients' suitability for group experience, abstinence during therapy, establishment of a strong positive transference, and, in a more general sense, the problem of keeping the group at a functioning numerical level in the face of frequent dropouts. These problems are common to all forms of group therapy, but, in addition, the alcoholic always manages to produce certain additional factors of his own. The treatment of alcoholics by group therapy in private practice is completely new, and therefore my suggestions are based primarily on personal observation.

My first endeavors, on meeting a new patient, are to

establish a strong beneficial father transference relationship. It is axiomatic that without this attempts at therapy will be futile. The alcoholic when he first meets the physician is either drinking or, alternatively, is in a withdrawal phase, and at this point inevitably begs for medical care to provide him with a good night's sleep, along with the most fervent of promises that he will never drink again and will follow to the letter the recommendations of the physician. Experience has shown that to believe this dooms management of the case. Such patients must be treated in an in-patient setting for a minimum of seven days, preferably in the wards of a general hospital. During the period of hospitalization several things are accomplished:

1. The patient is "dried out" and returned to a reasonable physiologic norm.
2. He is evaluated physically, psychologically and socially to determine the future therapy which would be most valuable for his particular type of alcoholism.
3. A strong positive transference relationship is developed between the physician and the patient. Confidence in the authority figure is somewhat restored and the beginnings of a breakthrough in his hostility barriers are accomplished by the use of psychotherapeutic agents and tender loving care.
4. Based upon the response to the above, a decision is made as to whether this particular subject is suitable for group therapy sessions. Should the evidence indicate that he is a potential candidate, then on the third or fourth hospital day an attempt is made to draw him into the group. Should he be resistant, however, to this idea, then the approach is modified and later office appointments are recommended for personal therapy combined with the use of vitamin B_{12}. One can then subtly plant the seeds of suggestion concerning the advantages of group therapy and hope that the patient will change his views on this subject.

The selection of patients for out-patient group therapy in private practice is based on several practical factors which are determined in two ways. A careful background history is taken while the patient is in hospital and he is requested to complete an interview questionnaire. This questionnaire has been carefully worked out to give the maximum information possible, both in a positive and negative sense, regarding the patient. Inherent in this form is an evaluation of the patient's sincerity, motivation, intellect, previous treatment, racial and hereditary background, as well as transportation problems should group therapy be recommended.

There is no doubt that in choosing patients for group therapy a prognostic evaluation is most important, but it is hard to give base lines for this and experience seems to be the only guide.

As was mentioned earlier, group therapy for the alcoholic has built into it certain unique problems. Primarily, of course, it is important to keep the patient sober for a sufficiently long period during therapy for him to acquire an insight into his basic problems. It is also his characteristic to resort to drinking again when group therapy shows that his basic problems are on the point of entering his conscious awareness. This process produces a constant and deep emotional pain, and with his weak ego, he is often unable to resist the return to the protection of the bottle. The therapist therefore has to be alert relative to each of his patients so that he can recognize when he is reaching this particular breaking point. Doctor Fox, of Florida, has stated that "The alcoholic is very fragile and should be handled accordingly." The usual non-directive type of group therapy can fail radically in the alcoholic and therefore must be frequently supplanted or replaced by strongly supportive and protective approaches. The therapist must, at all times, be ready to come to the emotional defense of his patient. It is certainly true that despite the most careful and alert observation of the patient, Antabuse® is still rather necessary to insure against a relapse into drinking.

Dropout is a vicious problem in group therapy for the alcoholic, even after careful screening and evaluation before allowing him to enter into the group. He tends to enter the therapy full of enthusiasm and vigor, to talk freely and to ventilate his problems in the first sessions, and, indeed, often will try to overwhelm the rest of the group with bizarre accounts of drinking and tales of fears, paranoid feelings and fantasies. He rapidly unburdens all his experiences of guilt and alcoholic activity, overextends himself and then fails to appear at the next session, or even worse, arrives fortified by alcohol in a hostile, aggressive and accusatory frame of mind. The alcoholic who enters group therapy with this approach rarely survives the first four sessions and derives no ultimate benefit. Such patients should be noted and every effort made to forestall the catastrophe by applying a strong directive hand and allowing other patients to take over the initiative. This, too, has its dangers since this new vigorous patient may feel rejected and fail to reappear at the next session. This brings one back to the importance of the transference dependency which the patient has developed towards the therapist, even prior to entering group therapy. Thus, where a strong dependency has been established and the father figure is prominent in his thoughts, his dependency needs are stronger than his feeling of rejection and he will continue to appear. The basic weakness in this whole approach to the alcoholic is that frequently the therapist has not had sufficient contact with the patient prior to his first group meeting to develop this type of dependency. When this is the case the patient rebels, returns to his alcoholic comforter and adds the therapist to the long list of authoritarian figures that he must both avoid and resist.

Mention was made earlier of the value of Antabuse in the early phases of group therapy. It should be borne in mind, however, that there have been reports of

severe physical and mental changes in both normal and schizophrenic subjects during Antabuse therapy. Certainly, the major psychotic exacerbations have been produced in those patients with a prior history of schizophrenia. The majority of my patients have, however, done well on Antabuse since a reduced dosage is customarily utilized. Tranquilizers may be helpful in preventing dropouts, since they allay tension and hostility during the primary phase of group therapy.

Homogenicity and heterogenicity are also a major problem in group therapy for these patients, particularly in view of their personal feelings of inadequacy and immaturity. Virtually any attempt to introduce an alcoholic into a heterogenous group results in failure. The patient's ego is not strong enough to cope with the normal group therapy patient. Thus it becomes necessary to initially form a homogenous alcoholic group and after cohesion has occurred to introduce gradually non-alcoholic group therapy patients until a balance is achieved. There is no doubt that the therapist who treats the alcoholic has major problems maintaining the integrity of his group. Hugh Mullens flatly asserted that he just had no success in keeping an alcoholic in his groups long enough to benefit the patient; while Alexander Wolf stated that in order to qualify for one of his groups, an alcoholic must have been sober for at least seven years.

The other characteristic so common to this group of patients, namely, poor interpersonal relationships can be dealt with effectively in long-term group experiences. The alcoholic has withdrawn from involvement with people and produced his own protective mantle. He stays warm within this little mental hut by the use of the bottle and suffers from the "lonely sickness." Should you ever have the opportunity to ask an alcoholic what he feels most, he will frequently state that it is the great overwhelming loneliness which can only be relieved by drinking. The alcoholic in group therapy resists personal involvement in as many ways as possible, but once this is overcome he realizes the extent of his problem and begins to interact and relate his own experiences in a meaningful fashion relative to other human beings. He discovers that his problems are not unique and begins to view them in the light of other people's experiences and gradually his loneliness disappears. Life begins to take on a new significance and alcohol gradually becomes unnecessary and finally is seen as a barrier to these newer and richer experiences of life. At this point, of course, life has become meaningful without alcohol. This can only be achieved if the alcoholic will stay with his group therapy for an adequate period of time so that the gaps in his personality formerly filled with alcohol can be replaced by more acceptable healthy human behavior patterns. Experience has shown that the patient who will faithfully and wholeheartedly accept group therapy for a period of six months or more will be materially benefited and frequently relieved of his alcoholic affliction.

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ANNUAL MEETING DATES FOR YOUR 1966 CALENDAR

Maine Medical Association, June 12-14, 1966 at The Samoset, Rockland, Maine

American Medical Association, June 26-30, 1966, Chicago, Illinois

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Flagyl eliminates the difficulties and frustrations that have long attended the treatment of trichomonal infection.

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In *women:* one 250-mg. oral tablet t.i.d. for ten days. A vaginal insert of 500 mg. is available for local therapy when desired. When the inserts are used one vaginal insert should be placed high in the vaginal vault each day for ten days, and concurrently two oral tablets should be taken daily.

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Pregnancy; disease of the central nervous system; evidence or history of blood dyscrasia.

Precautions and Side Effects

Complete blood cell counts should be made before and after therapy, especially if a second course is necessary.

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SEARLE

Research in the Service of Medicine

The Epileptic Brain and its Influence on History

EDWARD PODOLSKY, M.D.

Epilepsy has been an interesting riddle for hundreds of years. Although it is now known that there are several factors responsible for epileptic seizures, the chief cause is considered to be an irritated brain. The great majority of cases of epilepsy have been associated with various injuries to the brain, especially those which affect the parietal and frontal lobes. Tumors, scars, injuries, degenerative and atrophic changes have frequently been recorded as factors predisposing to epilepsy.

K. Winfield Ney had been very much interested in epilepsy for many years. It was his contention that the epileptic brain is always an irritated brain. There may not be any gross alteration in the structure of the brain, nor may there be any scars or other noticeable defects but a careful search will almost always show some sort of disturbance of the brain. In a group of 272 patients representing so-called idiopathic epilepsy, Ney found that there were various defects, small in themselves, but present nevertheless, which were responsible for the irritated brain and the convulsive seizures.

Arachnoidal defects or fistulas through which cerebrospinal fluid leaks into the subdural space have been found in practically every epileptic patient who presented no other defects. These fistulas are probably the result of tears in the arachnoid which have failed to heal. They are usually located at the extreme vertex of the brain where the strain of gravity is greatest.

It is the opinion of many authorities today that there must be some underlying anatomical factor responsible for the epileptic seizures. Considering the great weight of the brain, what maintains it in a state of postural stability within the cranial box? There are several factors, among which is the pressure of the cerebrospinal fluid. Also, there are various strands of tissue which help to maintain the brain in a smooth, calm milieu. Any factor which causes an escape of cerebrospinal fluid of the brain, or which causes tears in the fine strands of tissue, at once abolishes the normal postural stability of the brain. Irritative factors then come into play, and epilepsy becomes manifested.

Epilepsy and leadership have often gone hand in hand down through the ages. Many of the great of mankind had irritated brains and epileptic incidents at regular intervals, T. C. von Storch points out that St. Paul, Mohamet, Luther, Loyola, Dante, Petrach, Pascal, Schumann, Richelieu, Byron, Paracelsus, Empodocles, Beethoven, Flaubert and Alexander the Great, were but a few of the great who suffered from epilepsy. So did Joan of Arc, and Emanuel Swedenborg. W. G. Lennox adds the following to the list: Peter the Great, Louis XIII of France, Charles V of Spain, Alfred the Great, William III of England, Guy de Maupassant, Hector Berlioz, Blaisem Pascal.

In the field of poetry Lord Byron is an outstanding example of the fact that epilepsy does not cloud a man's mentality. There has rarely been written poetry of greater brilliance, passion and beauty as that shown in the works of Lord Byron. An accomplished artist of the letters, he stands in high esteem throughout the civilized world and his writings have been translated into all major languages.

Arthur Wellesley Wellington was one of the greatest military leaders and statesmen of all times. He is said to have suffered from attacks of epilepsy during his adult life. His brilliant campaigns and eventual victory over Napoleon made him a national hero in Great Britain, admired throughout liberated Europe.

The French actress Rachel, one of the most celebrated artists of the stage, was subject to fits of an epileptic character. Rachel's best known roles consisted of the impersonation of evil passion which she presented with such majestic dignity that it made even the most repelling character fascinating and respected. She was as successful in guest appearances in London as she was on the French stage.

The famous English poet, Algernon Charles Swinburne, who began to show signs of epilepsy at the age of 25, is a prominent example that despite epilepsy a man may develop to true greatness. His poems, ballads and songs contain some of the most beautiful lyrics ever written in any language. Swinburne's extraordinary metrical ingenuity, the vigor and verve, harmony and charm of his poetry have been admired by millions throughout the world.

The Dutch painter, Vincent Van Gogh, one of the masters of Post-impressionism, is said to have suffered from the psychic equivalent of epilepsy during his adult life. Although Van Gogh had great difficulty in social adjustment, he achieved universal fame as one of the founders of modern art.

The brilliant Italian virtuoso and composer, Niccolo Paganini, is said to have suffered from epileptic seizures. His matchless masterful performances–especially his unprecedented playing on the fourth string alone–as well as his beautiful vivacious compositions brought him triumphant success and fame throughout the world. Paganini was admired by his great musical contemporaries as much as he was loved by his audiences.

Dostoevski also was a victim of epilepsy. This melancholy Russian literary giant left the most eloquent description of epilepsy yet to be found. In his novel, "The Demons" occurs the passage.

"There are moments–and it is a matter of five or six seconds–when you suddenly feel the presence of eternal harmony. This phenomenon is neither terrestrial nor celestial, but it is an indescribable something which man, in his mortal body, can scarcely endure–he must either undergo a physical transformation or

die. It is a clear and indisputable feeling: all at once you feel as though you were placed in contact with the whole of nature, and you say, 'Yes, this is true.' When God created the world He said at the end of every day of creation: 'Yes, this is true! This is good!' And it was not tenderness, nor yet joy. You do not forgive anything, because there is nothing to forgive . . . neither do you love . . . no, this feeling is higher than love. The terrible thing is the frightful clearness with which it manifests itself, and the rapture with which it fills you. If this state were to last more than five seconds, the soul could not endure it, and it would have to disappear. During the five seconds, I could live a whole existence, and for that I would give my whole life and not think I was paying too dearly."

Many of the great military geniuses were men with irritated brains. Alexander, Julius Caesar and Napoleon were epileptics. The Roman historian Suetonius described two of Caesar's epileptic seizures. At Thapsus, says Dr. Pirkner, Caesar had premonitory symptoms, and "shaken with tremblings and feeling the aura had barely time to be carried into a tower, where the seizure overcame him. The premonitory symptoms were tingling and trembling lodged in one of his feet, which subsequently he felt rising up the whole of his body, and when they reached his head, he lost consciousness."

Napoleon had epileptic seizures on occasion. As reported by MacLaurin, Napoleon in September, 1805, was seized with an attack, fell to the ground and struggled convulsively. This constant cerebral irritation probably led him on to greatness. Inner compulsions that can not be stayed caused some men to reach out beyond a point where a man with a calm brain would not dare to venture.

The great religious leaders were also men with seething brains, irritated by something within the brain that disturbed its equilibrium. Moses was an epileptic. So was Mohamet, the founder of Islam. Says Dr. Pirkner, "Mohamet would be seized with violent trembling, followed by a kind of swoon, or rather convulsion, during which the perspiration would stream from his forehead in the coldest weather; he would lie with his eyes closed, foaming at the mouth and bellowing like a young camel."

St. Paul, the apostle who disseminated the Christian doctrine, was another of the world's great spiritual leaders who suffered from epilepsy. It is the belief of modern neurologists that his constant drive was actuated by an irritated brain, or at least by an unstable brain (from the physical point of view).

To analyze each of the foregoing cases of epilepsy with any degree of thoroughness would take us beyond the space available for the present discussion. It will suffice to take one celebrated epileptic and discuss his case in some detail. Our choice is Gustave Flaubert, one of the world's great men of letters.

Flaubert was twenty-two years old when he had his first epileptic seizure. It was in the month of October, 1843. Other attacks followed; four of them in two weeks. These seizures all occurred in the same way and were preceded by the same symptoms. Suddenly, without apparent reason, he would turn very pale and with an expression full of anguish, would shrug his shoulders in a heart-rending gesture of discouragement, saying, "I have a flame in my right eye; everything seems golden color to me." This strange condition would continue for a few minutes, until becoming still paler with a desperate expression on his face he would run toward his bed and stretch himself out on it as though he were lying down in a coffin. Then with a cry he would pass into the throes of convulsion. The paroxysm would be succeeded by a deep slumber.

Gustave Flaubert was a typical genius with an irritated brain. To treat things lightly, to put his loves or his hatreds into perspective was impossible for him; his method was always one of excess. It was in depicting indignations that his eruptions of violence and of paradox found their best expression.

Violence and excess are the only traits of character of which this style bears the imprint. The hypersensitivity of the epileptic is equally marked. This almost feminine quality sometimes brought tears to his eyes but more often it rendered him irritable or irascible, ready to fly into a rage for anything or nothing at all. When writing, the search for a word would throw him into a state of anger and he would thump the table with his fists. He would pass from the highest expectation to the deepest depression with greatest rapidity and without apparent cause.

Flaubert suffered most of all in connection with rhythm and rhyme. Bouilhet stated: "There is a curse upon him; he is a lyric poet who cannot write a verse." Flaubert writhed in his powerlessness. "And all the time I feel it inside me," he cried, referring to the beat of the rhythm that escaped him.

"There comes a moment when one feels a longing to make oneself suffer when one hates one's own flesh and wants to throw mud in one's own face," Flaubert declared. At the same time he said, "I love my work with a frenzied and perverted love, as an anchorite loves the hair cloth that tortures his body." Flaubert was a sadist, and a strong touch of sadism is evident in much of his work. This characteristic is quite common among epileptics. Flaubert's favorite type of hero, grotesque, coarse, sententious, violent, obstinate and patient, is the model of the author.

The epileptic is not necessarily a physically incapacitated individual. Nor is he constantly in the throes of convulsions. There are those cases of sub-clinical epilepsy which are seizure-free. Many such individuals are chronically in a state of emotional tension which puts them on edge, full of drive and activity. In order to obtain release from this chronic tension they must work beyond their capacities. Many high-powered business executives, writers, artists, composers are of this variety. The irritated brain serves as the impetus to productive activity.

The Aged Patient

Overtreated And/Or Undertreated†

SIDNEY R. BRANSON, M.D.*

One need not be a physician to be aware of the increase in the number of elderly people in our country today. Due to progress in medicine and surgery, more people are living to so-called ripe old age. In spite of this increase in life expectancy, people do reach the point where the ravages of arteriosclerotic disease neoplasms and accidents begin to take their toll. These people now fill the active wards of most hospitals and nursing and convalescent homes.

Our nursing homes today are a far cry from the prewar "Old Peoples' Homes" or Town Farms. Today they serve as annexes of large general hospitals, staffed with RN's, practical nurses and nurses' aides. Physicians make periodic visits and most of the elderly get good medical care.

Two of our nursing homes in Windham have large day rooms where ambulatory patients gather and sit and gossip, play cards, or have fights over who took the magazines; there are even a few flirtations. The folks feel they are not abandoned and left to wait for death to come. They are stimulated; they think of their appearance, try to be neat. They have a purpose in life.

This happy situation is augmented by a friendly, breezy, happy nursing staff, nurses who cheer up their patients, do their hair. make them laugh and encourage them to eat. This means a great deal to the old folks who are alone. away from family and friends. They may feel miserable because of "strokes", rheumatism, indigestion, old hip fractures, colostomies, parkinsonism or amputations.

On the other hand, I know of very fine, clean and efficient nursing homes which unfortunately have the atmosphere of a mortuary or museum. The patients stay in their cold and quiet rooms; there is no communication, no hum of conversation–just a stillness. Each patient is compartmentalized, lying in bed or sitting in his wheelchair in his room, awaiting the grim reaper.

The doctor should not neglect this segment of his practice. Nursing home patients should be visited once a week regularly and emergencies or sudden illness treated as any occur. In making rounds, see the ones who have problems, also say a few words or shake hands with the well patients. This gives them the feeling they are not abandoned and that the doctor is interested in them.

Let us look at some of the more common problems of the aged, see what is generally done or not done for them and then attempt to establish a medical philosophy to cover this important segment of practice. We must not forget that geriatrics encompasses every specialty but pediatrics and the problem of the aged concerns us all.

The largest group of diseases involving old people is the arteriosclerotic syndrome with all its ramifications and complications. Heart disease and strokes seem to take the lions share in this broad area. For heart disease, we have anticoagulants, digitalis, diuretics, vasodilators, etc. For strokes we have bed rest plus the newer surgical techniques and various rehabilitation and function restoring measures. With so much potential therapy available today, no one should be neglected. There are still some doctors who do not use antihypertensive drugs for blood pressure; who have a fatalistic attitude toward the hemiplegic who say "You're paralyzed and there's nothing to do about it;" who simply tell a patient their condition is due to old age and there is little worth doing. I believe in encouraging a patient under all circumstances, even when you have private reservations about how much good a projected therapy will accomplish.

On the other hand, too often in so-called hopeless cases, many painful and costly procedures are being carried out. An elderly person will have a shock and be taken to the hospital in an unconscious state; the patient is in deep coma and obviously moribound, but what is in store for him: spinal taps, EKG, numerous blood chemical tests, x-rays, intravenous therapy and fluids, and consultations, yet everyone realizes the prognosis is very poor, a matter of a few hours or a few days. As a matter of fact our professor of neurology used to say that the stroke cases on the charity wards did better than the wealthy stroke patients. The former were put to bed in the large wards and were left alone where as the latter had frequent consultations and ministrations by various personnel and private duty nurses.

Neoplasms account for a large share of morbidity and mortality in the aged. Here the guide lines are well established–make the diagnosis and attempt the cure.

Age makes little difference to the surgeon today, with proper knowledge of anaesthetics, fluid balance, etc. In one of our nursing homes, we have two elderly ladies with colostomies for carcinoma of the bowel both of over ten years' duration. In fact one of them fell and broke her hip last winter and it was repaired in this hospital and she's up and about every day now. We have one elderly lady about 88 who developed facial car-

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†Presented at Seventeenth Annual Alumni Day, Maine Medical Center, Portland, Maine.

*Attending physician, Department of General Practice, Maine Medical Center, Portland, Maine.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Well-Known Public Health Officials Retire

In the closing months of 1965, two key officials of the Maine Bureau of Health–pioneers in their respective fields of public health resigned to accept well-earned retirement. They are Dr. Elmer W. Campbell, long-time head of the State's Division of Sanitary Engineering and one of the first academically qualified doctors of public health in Maine, and Miss Ruth Thorndike Clough, member of the well-known medical family of Bangor who, in more recent years, pioneered in health education within and without the Department.

Each has authored numerous professional papers in this section of The Journal since the Departmental page was established in 1956.

In each case, retirement was voluntary. Miss Clough in recent years has made her home at Damariscotta with two Departmental colleagues, Miss Katherine Gay, recently retired administrative assistant in the Division of Tuberculosis Control and the latter's successor, Miss Clara Floyd. Dr. Campbell and his wife plan a trip to Florida where they have a winter home, but will be spending most of their retirement years in Maine.

A group of private and public health agency officials, Maine Safety Conference leaders and Departmental personnel honored Miss Clough at a luncheon in connection with the closing session of the annual Safety Conference at Poland Spring in September. She was presented with an electric typewriter which, her friends report, is already being put to good use at Damariscotta.

On December 15 at the Augusta House, 175 associates and state and local officials honored Dr. Campbell at a retirement luncheon. The Commissioner, Dean Fisher, M.D., presided as toastmaster and a letter of testimonial and good wishes was read from Governor John H. Reed. Mayor Sylvio Gilbert of Augusta was among the head-table guests. Dr. Campbell was presented with a Polaroid camera with all attachments. He completed his final day as head of the Sanitary Engineering Division on December 17.

During his long career in public health, Dr. Campbell a Maine native had numerous "firsts". Immediately upon graduation from Colby College in the war-time year of 1917, he attended Naval Officer Material School and was honorably discharged as an Ensign in the U.S. Navy. After one and one-half years at the Massachusetts Institute of Technology, he transferred to the Harvard Technology School of Public Health, graduating with a Certificate of Public Health in 1921. He then went on to the University of Michigan where he received his degree of Doctor of Public Health in 1923. He later received an honorary degree as Master of Public Health from Harvard.

Dr. Campbell began his long tenure as chief sanitarian for the State immediately following graduation from the University of Michigan, a position he filled with distinction 42 consecutive years, except for leave of absence in World War II. A Navy officer in the first World War, he served as an Army officer in the Pacific in World War II and retired as a Major in the Reserve.

One of Dr. Campbell's outstanding achievements was his work in securing passage of the first State regulatory laws in cosmetics, a measure that was taken to the U.S. Supreme Court and affirmed. His records were used as the basis for important changes in the U.S. Food-Drug and Cosmetic laws.

Another milestone was in 1950 when he succeeded in developing the first Bangs Disease Testing Program which resulted in Maine becoming an accredited free area. Still another was his organization of the Maine Water Utilities and also the Maine Municipal Association of which he was the first president.

Dr. Campbell served as Mayor of Hallowell for eight years and has been active in various civic groups as well as professional societies both State-wide and nationally. He is a Life Fellow of the American Public Health Association and was honored by that organization for more than 40 years of membership, receiving a certificate paying tribute to "such long sustained affiliation–an inspiration to the public health profession".

He was one of the charter members of the Augusta Rotary Club–served as Secretary and President with an outstanding attendance record and service.

Dr. Campbell is the author of numerous papers in regional and national public health journals.

RUTH CLOUGH

It is rather ironic that someone who wrote with such facile grace about others and their public service as did Ruth Clough should have only a factual (although ad-

miring) biographer to outline her numerous accomplishments in the relatively new field of public health education in Maine.

A Bangor native, daughter of the late Dr. and Mrs. Herbert T. Clough, she followed an early interest in education by a career in Maine politics, followed by still another in public health education. She was graduated from Goucher College in 1925 and subsequently earned graduate degrees from the Columbia University School of Business Administration and the University of Maine as well as her degree as Master of Science in Hygiene from the Harvard School of Public Health in 1956 after joining the Departmental staff in 1949.

Miss Clough was long active in Penobscot County organizations including the Bangor-Brewer TB and Health Association. Before joining the Departmental staff she was for five years a member of the Health and Welfare Advisory Committee.

She served 10 years in the Maine Legislature, two terms in the House and three as a State Senator from Penobscot County with service on key legislative committees including Public Health.

There were many highlights in her 15 years of service as Health Education Consultant for the Department and included must be two highly successful week-long sessions of the New England Health Institute at Colby College which she planned and directed colleagues and private agency people to implement. Still another was the first International Conference on Mental Retardation which brought to Maine some 600 physicians and other experts in that field from all over the world.

Working closely with the Maine Medical Association she was largely responsible for the success of numerous public health campaigns, among them the Maine Diabetes Testing Program, the State-wide eye testing program for pre-school children and numerous others over the years.

Miss Clough was elected President of the New England Health Education Association and was an active member of various other professional organizations.

THE AGED PATIENT, OVERTREATED AND/OR UNDERTREATED – Continued from Page 16

cinoma. Being bedridden, she was sent by ambulance to a dermatologist who treated her in his office and sent her back cured.

Accidents and traumatic experiences are common among the elderly. The hip, wrist, and humerus are common fracture sites. Here, too, age is no bar to adequate treatment.

Upper respiratory infections and the "flu" are often troublesome and may spread through a nursing home by leaps and bounds. We believe in the generous and prolonged use of antibiotics. It seems the infections hang on longer in the older patients; sometimes we keep up therapy for two or three weeks, preferring to use the broad-spectrum variety of drugs.

Skin care is a big problem, especially in the bedridden patient. Staphylococcus infection and bed sores can be stubborn and many times the treatment can only be symptomatic and palliative at best.

Last, but not least, are the neuropsychiatric problems. Insomnia, depression, senile psychosis and mere senility seem to be ever-present and probably the most distressing to the patients and the attendants. A warm and happy atmosphere is good therapy. For the restless and the irritable Thorazine® in adequate doses is often helpful. Placebos also work well in many cases.

In summary, the care of the aged should be the same as that given to any other group of patients. The physician should not rationalize and deny therapy because someone is very old and it's his fate to be ill. Youth is impatient with oldsters, but as the doctor reaches middle age, he is likely to be more tolerant and understanding of the older folks and their problems.

There is a sadness about old people but also a beauty and dignity. The physician should strive to help, comfort and encourage this group of patients, and in return will find them responsive and grateful.

797 Gray Road, South Windham, Maine

Maine Heart Association Notes
116 State Street, Augusta, Maine

Intestinal Infarction In The Absence Of Occlusive Mesenteric Vascular Disease

"Although infarction of the bowel in the absence of mesenteric vascular occlusion has been reported many times . . ., this entity has apparently only slowly made its way into clinical consciousness. It is known to occur in the aged and chronically ill, is frequently associated with congestive heart failure, myocardial infarction, arrhythmias and hypotension and may present as an abdominal catastrophe or simply as irreversible shock. . . . Congestion of the viscera is a common finding, and stagnant anoxia has been suggested as a cause of the tissue damage. . . . More recent studies have demonstrated mesenteric angiospasm in many of the underlying cardiovascular conditions, and the resulting shunting of blood and reduction in mesenteric blood flow has been suggested as a cause of the bowel ischemia and necrosis.

"Nine cases of intestinal infarction in the absence of occlusive mesenteric vascular disease are . . . reported. . . . The histologic examination of the bowel . . . revealed edema, vascular congestion and hemorrhage into the mucosa and submucosa. Some degree of necrosis of the bowel wall was observed in all. . . . Evidence of heart disease was present in all cases with congestive heart failure, present clinically in 7 of the 9 cases, and pathologic and anatomic evidence of congestive failure demonstrated in all.

"Of the total of 109 cases now reported 75 had congestive heart failure. Most of these patients had such cardiac or circulatory conditions that a reduced cardiac output and mesenteric angiospasm might be anticipated. That angiospasm . . . can contribute to the ischemia and necrosis of the bowel . . . appears reasonably well established. In addition, it also seems reasonable to presume that the vascular sclerosis in many of these patients might also be an underlying factor."

Submitted by Jacob B. Dana, M.D.

Reference: Kligerman, M. J. and Vidone, R. A. The American Journal of Cardiology, Volume 16, pages 562-570, 1965.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Smoking and Doctors

The American Cancer Society went on record in October, 1965 in terming the physician the key figure in reducing deaths caused by cigarette smoking.

The Society's Board of Directors at conclusion of its 52nd annual meeting in New York urged physicians to recognize the need for stopping smoking as an example to patients and teenagers.

In a resolution which reviewed the position of state and national medical associations on cigarette smoking, the Board stated, "It is thus safe to say that the view of the overwhelming majority of the medical profession and other groups concerned with health favors action to reduce the damage leveled by cigarette smoking."

The resolution said the physician is "recognized as the most significant factor in the nation's efforts to reduce death and disease related to cigarette smoking."

The doctors were called upon to participate with their professional organizations in attempting to reduce cigarette smoking and "personally to instruct those in their care about the health hazards of cigarette smoking."

The American Cancer Society also asked that physicians devote time to telling young people about the dangers of cigarettes with "particular emphasis on the difficulties of breaking the cigarette smoking habit, once it is firmly established."

The Board's resolution concluded, "Resolved: That the physician recognize the importance of his attitude and action as a non-cigarette smoker in setting an example before those he treats and teenagers whom he attempts to advise."

County Society Notes

LINCOLN-SAGADAHOC

The annual meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on October 19, 1965. The meeting was called to order by the Secretary, George W. Bostwick, M.D.

The following members were elected to serve as Delegates and Alternates to the Maine Medical Association House of Delegates:

Delegates: Drs. John F. Andrews, Boothbay Harbor and Ralph C. Powell, Damariscotta. Alternates: Drs. Paul A. Fichtner and Charles E. Burden, both of Bath.

Mr. Richard F. Nellson of the Associated Hospital Service of Maine was introduced by John F. Dougherty, M.D. Mr. Nellson spoke on the implications to physicians and dentists of the new Federal Medicare plans under Social Security.

The Lincoln-Sagadahoc County Medical Society met at The Ledges in Wiscasset, Maine on November 16, 1965. The meeting was called to order by the President, Carl R. Griffin, Jr., M.D.

Dr. Griffin introduced Mr. Richard F. Nellson, Executive Director of the Associated Hospital Service of Maine, who spoke on the choosing of a fiscal intermediary for Plan B of the Medicare program. Mr. Nellson felt that the Associated Hospital Service of Maine could do the best job as fiscal intermediary of any of the candidates for the appointment.

Francis A. Winchenbach, M.D., Chairman of the Maine Medical Association's Health Insurance Committee, requested action on the county society's choice for a fiscal intermediary. Paul A. Fichtner, M.D. moved, and John F. Dougherty, M.D. seconded, that this society endorse the choice of the Associated Hospital Service as fiscal intermediary for Plan B.

Dr. Richard J. Martin, Chief Medical Officer of the Brunswick Naval Air Station, spoke on the Naval Flight Surgeon and showed a training film.

GEORGE W. BOSTWICK, M.D., *Secretary*

CUMBERLAND

Seventy-six members and guests were present at the meeting of the Cumberland County Medical Society which was held at the Old Westcustogo Inn in Yarmouth, Maine on October 21, 1965.

Alvin A. Morrison, M.D., President of the society, presided. The Program Chairman, Charles R. Glassmire, M.D., introduced Mr. Jotham Pierce, chairman of a panel on estate planning. Other members of the panel were Mr. Leon Stover and Mr. Frank Helyar.

Dr. Morrison announced that the Executive Committee had written a letter to Mr. Graham Watt, the City Manager of Portland, urging that the vacancy caused by the resignation of Boris A. Vanadzin, M.D. be filled by a physician and offering help of the Cumberland County Medical Society in finding a replacement for Dr. Vanadzin and in providing professional help during the interim.

The resolution on the death of Howard R. Ives, M.D. was read. The resolution on the death of Jack Manol, M.D., which was previously presented at the Maine Medical Center, was accepted in behalf of the Cumberland County Medical Society.

Dr. Morrison requested a report at the next meeting from the committee investigating the unified hospital complex in Portland.

A meeting of the Cumberland County Medical Society was held in the Castine Room of the Sheraton-Eastland Motor Hotel in Portland, Maine on November 18, 1965 with eighty members and guests present.

Following a social hour and dinner, the introduction of guests, and the secretary's report, it was announced that Dr. John T. Kennedy, Jr. is moving to Wisconsin and that Dr. Richard A. Marshall is moving to Lewiston.

Douglass C. Pennoyer, M.D. reported that little progress had been made in his committee's investigation of the possibility of a unified medical complex for Portland, despite national interest and some local support of the concept. He indicated that this is not a "dead" issue but that its implementation is far from a reality. George F. Sager, M.D. reported on progress by the Medical-Legal Liaison Committee and promised further definitive proposals upon which positive action should be taken. Dr. Morrison appointed a nominating committee with Dr. Charles R. Geer as Chairman and with Drs. G. Hermann Derry and Donald F. Marshall as members. Dr. Morrison reported on a meeting with Mr. Graham Watt concerning the possibility of obtaining federal funds to be used for a teacher of public health at the local hospitals who could also serve as Director of Public Health for the City of Portland.

Albert Aranson, M.D. discussed the meeting of the Health Insurance Committee which was held on October 31, 1965 at which time the nomination of a carrier under Part B of Medicare was discussed but referred back to the county societies for instruction. He announced that the "BSB" Blue Shield Contract would be dropped and a new $7,500 level plan would be developed with payments based upon prevailing fees rather than a fixed schedule. Social security coverage for physicians was briefly discussed, as were utilization committees and the possibility of a retirement plan utilizing deferred payment of Blue Shield fees.

STANLEY B. SYLVESTER, M.D., *Secretary*

KENNEBEC

A meeting of the Kennebec County Medical Association was held at the Jefferson Hotel on October 21, 1965. John F. Dougherty, M.D., President of the Maine Medical Association, was an unexpected visitor. Dr. Dougherty spoke briefly and was mainly concerned with the Maine Medical Education Foundation. He expressed the hope that all those who have not as yet supported the fund would do so at their earliest convenience.

A letter was read from Price A. Kirkpatrick, M.D. explaining a seminar being sponsored by the Kennebec Mental Health Clinic to begin in November for the benefit of any of the medical profession within the county who might be interested.

William E. Schumacher, M.D. discussed the wider ramifications of the Medicare bill which must be considered in the near future if the program is to be utilized to the fullest extent. This was in regard to Title 19 of the law to provide total medical care for the medically indigent as well as those eligible under the Social Security system.

Burton L. Olmsted, M.D., an Associate Surgeon at the Maine Medical Center, was introduced by the President, John D. Denison, M.D. Dr. Olmsted discussed present methods of plastic surgery and illustrated his talk with specific cases.

The Kennebec County Medical Association met at Togus, Maine on November 18, 1965, following a dinner served at the Veterans Administration Center.

The President, John D. Denison, M.D., gave a brief report of the most recent meeting of the Insurance Council and asked that the membership be considering whom they might elect to be a fiscal intermediary for the Medicare program.

Alta Ashley, M.D. gave a brief follow-up on the Diabetic Detection Week program. Robert L. Ohler, M.D., Chief of the Medical Service at the Veterans Administration Center, explained a research program which is now underway there

and asked that any veterans eligible for care at the Center who appear to be suffering from incipient "d.t.'s" be referred to the Center for possible inclusion in the research program.

Dr. Arthur A. Sasahara, Assistant Chief of the Medical Service and Director of the Cardiopulmonary Laboratory at the Veterans Administration Hospital in West Roxbury, Massachusetts, presented the clinical program. Dr. Sasahara spoke on "Pulmonary Thrombo-Embolic Disease." His presentation was a clear, concise and well illustrated presentation of the diagnostic studies, the management, and the end results of this distressing disease.

EARLE M. DAVIS, M.D., *Secretary*

Book Review

Cardiomyopathies. Ciba Foundation Symposium, Edited by G. E. W. Wolstenholm, O.B.E., M.A., M.B., F.R.C.P. and Maeve O'Connor, B.A. Published by Little, Brown and Company, Boston 1964. 428 Pages. Price $12.50.

This delightful three-day symposium describes primarily the syndrome of obstructive cardiomyopathy. Many of the world's leading authorities discuss the clinical recognition, pharmacological, and radiological findings in obstructive cardiomyopathy. Readers will enjoy the clarity and vigor of each section, which includes much controversy.

Even the experienced reader will find many new stimulating data, such as the electron microscopic and histochemical studies of cardiomyopathic muscle. These techniques demonstrate a marked proliferation of sympathetic nerve fibres, marked increase in the number of mitochondria, shortness of individual myofibrils, increased number of lysosomes. Various muscle enzyme abnormalities are discussed. Digitalis and isoproterenol intensify the obstruction to left ventricular outflow, while pronethalol (Nethalide) a beta-adrenergic blocking drug relieves it.

There is much thought provoking discussion concerning the possible pathogenesis of the obstructive type of cardiomyopathy, including a suggestion that early activation of the septal or basal region of the heart may play an important role in its production. Sudden death appears to occur much more frequently in patients with the familial form of the disease who are asymptomatic.

The last day of the three-day symposium is an excellent but short review of other types of cardiomyopathy including endomyocardial fibrosis, fibroelastosis, Friedreich's disease, and viral myocarditis. The discussion of viral myocarditis is especially valuable. It is unfortunate that the symposium could not have lasted another three days to cover these conditions in as much detail as it covered the obstructive cardiomyopathies.

This book is very well planned and has a high standard of excellency. It must be read several times, as it is intricate and full of controversy. A good background of physiology is assumed by the editors. The illustrations are of good quality, and there is a short, adequate bibliography and index. This book is highly recommended to internists, cardiologists, and surgeons.

PETER F. LANSING, M.D., Augusta, Maine

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Precautions: *Meprobamate*—Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage.

Benactyzine hydrochloride—Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria.

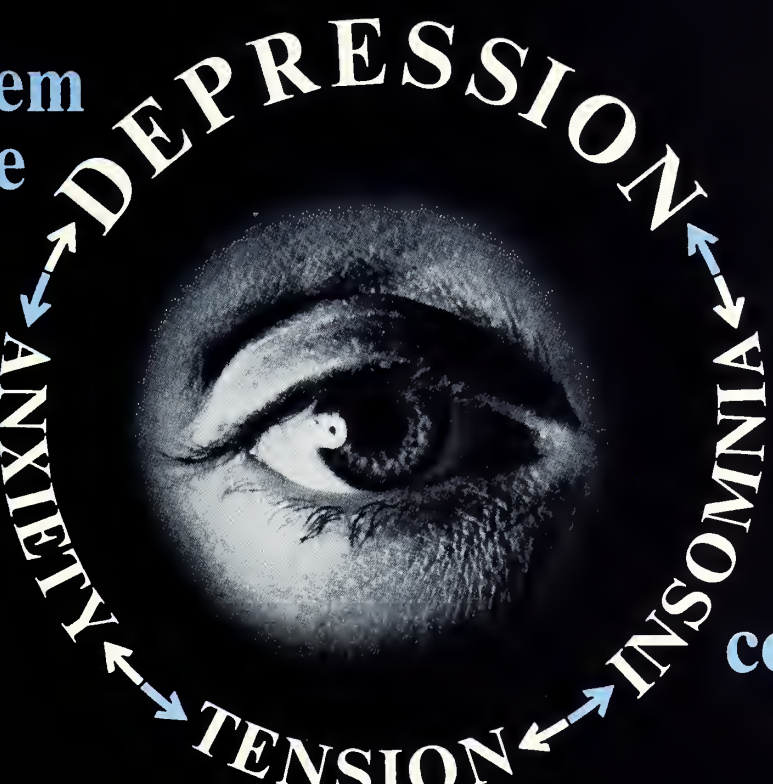
Meprobamate—Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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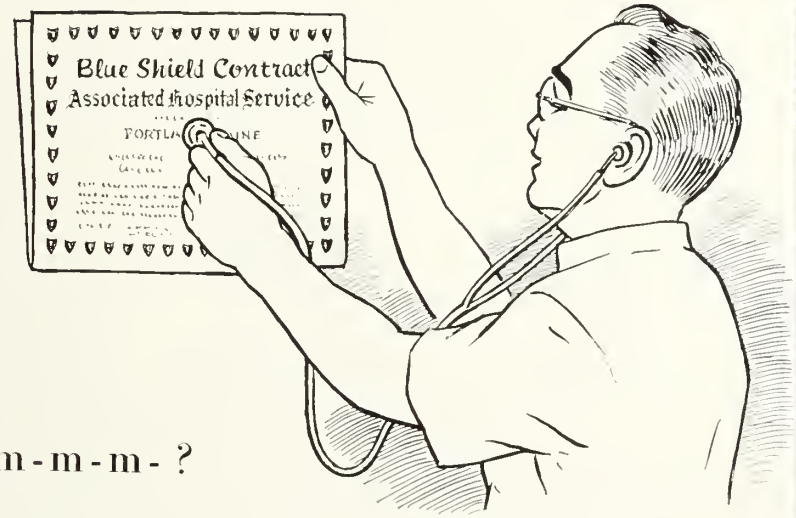
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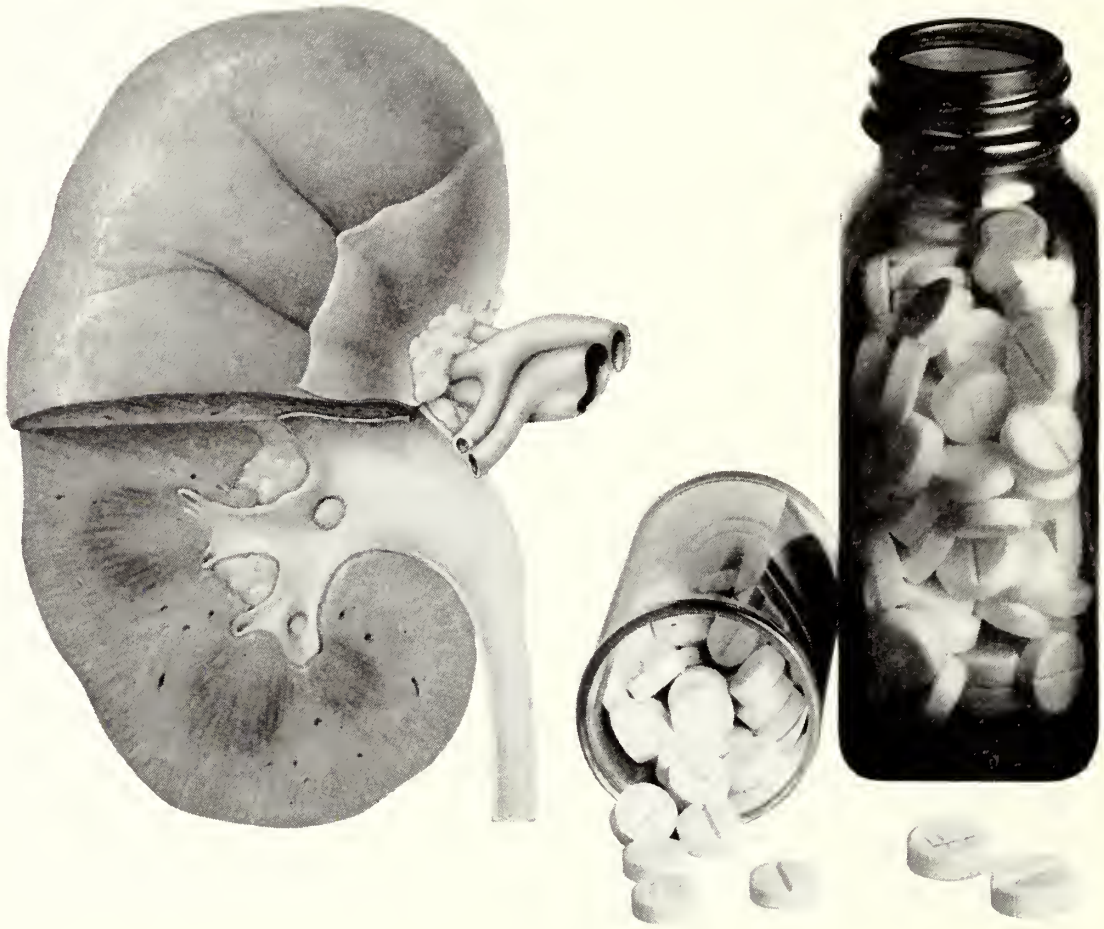
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1. Torosdag, S., Schvartz, N., Fletcher, L., Fertig, H., Schwartz, M. S., Quan, R. F. B., and Bryant, J. M., Pargyline Hydrochloride as an Antihypertensive Agent With and Without A Thiazide, Am. J. Cardiol., 12:822, Dec., 1963.

2. Pollack, P. J., Pargyline Hydrochloride and Methyclothiazide Combined In The Treatment of Hypertension, Cur. Thera. Res., 7:10, Jan., 1965.

3. Bryant, J. M. et al., Antihypertensive Properties of Pargyline Hydrochloride, New Non-Hydrazine Monoamine Oxidase Inhibitor Compared with Sulphonamide Diuretics, J.A.M.A., 178; 406, Oct., 1961.

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BP reductions in the recumbent and sitting positions often are nearly as great as in the standing. In clinical trials, the average *recumbent* BP reduction was 36/18 mm. Hg.



The average *standing* reduction in clinical trials was 45/22 mm. Hg. Thus the difference between the standing and recumbent readings was only 9/4 mm. Hg.

Significantly lowers blood pressure in all body positions; less likelihood of orthostatic hypotension

In clinical trials, the average reduction in standing blood pressure was 45/22 mm. Hg.; in the sitting position it was 48/20 mm. Hg.; and in the recumbent position, 36/18 mm. Hg.

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Prescribing information for **EUTRON**

INDICATIONS: Eutron (pargyline hydrochloride and methyclothiazide) is indicated in the treatment of patients with moderate to severe hypertension, especially those with severe diastolic hypertension. It is not recommended for use in patients with mild or labile hypertension amenable to therapy with sedatives and/or thiazide diuretics alone.

CONTRAINDICATIONS: Eutron is contraindicated in patients with pheochromocytoma, advanced renal disease, paranoid schizophrenia and hyperthyroidism. Until further experience is gained it cannot be recommended for use in patients with malignant hypertension, children (under 12 years of age), or pregnant patients.

The concomitant use of the following is contraindicated: other monoamine oxidase inhibitors; parenteral forms of reserpine or guanethidine; sympathomimetic drugs; foods high in tyramine such as cheese; imipramine and amitriptyline, or similar antidepressants; methyldopa. A drug-free interval of two weeks should separate therapy and use of these agents.

WARNINGS: Pargyline hydrochloride is a monoamine oxidase inhibitor. Patients should be warned against eating cheese, and using alcohol, proprietary drugs or other medication without the knowledge of the physician. When it is necessary to administer alcohol, narcotics (notably meperidine), antihistamines, anesthetics, barbiturates and other hypnotics, sedatives, tranquilizers, or caffeine, these agents can be used cautiously at a dosage of ¼ to ⅕ the usual amount. Avoid parenteral administration where possible. Withdraw pargyline two weeks before elective surgery.

Patients should be warned about the possibility of postural orthostatic hypotension. Those with angina or other evidence of coronary disease should not increase physical activity. Pargyline may lower blood sugar. Potassium depletion is unlikely at the recommended dosage, but if it occurs, adjust dosage or withdraw or provide added natural food sources of potassium; potassium tablets should be avoided wherever possible, as bleeding or obstructive ulceration of the small bowel has been associated with their use; potassium levels should be especially watched if the patient is on digitalis or steroids, or if hepatic coma is impending.

PRECAUTIONS: When determining the antihypertensive effect of Eutron, blood pressure should be measured while the patient is *standing*. Use with caution in hyperactive or hyperexcitable persons. Such persons may show increased restlessness and agitation. Withdraw drug during acute febrile illness. Watch patients with impaired renal function for increasing drug effects or elevation of BUN and other evidence of progressive renal failure; withdraw drug if such alterations persist and progress. Pargyline has not been shown to cause damage to body organs or systems. As with all new drugs, complete blood counts, urinalyses, and liver function tests should be performed periodically. The drug should be used with caution in patients with liver dysfunction. With prolonged therapy, examine patients for change in color perception, visual fields, and fundi.

Elevated blood urea nitrogen, serum uric acid or blood sugar are possibilities attributable to the methyclothiazide in Eutron. Methyclothiazide may also reduce arterial response to pressor amines. Blood dyscrasias, including thrombocytopemia with purpura, agranulocytosis and aplastic anemia, have been seen with thiazide drugs.

SIDE EFFECTS: The use of pargyline may be associated with orthostatic hypotension. Mild constipation, slight edema, dry mouth, sweating, increased appetite, arthralgia, nausea and vomiting, headache, insomnia, difficulty in micturition, nightmares, impotence, delayed ejaculation, rash, and purpura have been encountered with pargyline. Hyperexcitability, increased neuromuscular activity (muscle twitching) and other extra-pyramidal symptoms have been reported. Drug fever is extremely rare. Congestive heart failure has been reported in a few patients with reduced cardiac reserve. Nocturia has been observed with the combination. If side effects persist, despite symptomatic therapy or reduction of the dose, discontinue the drug.

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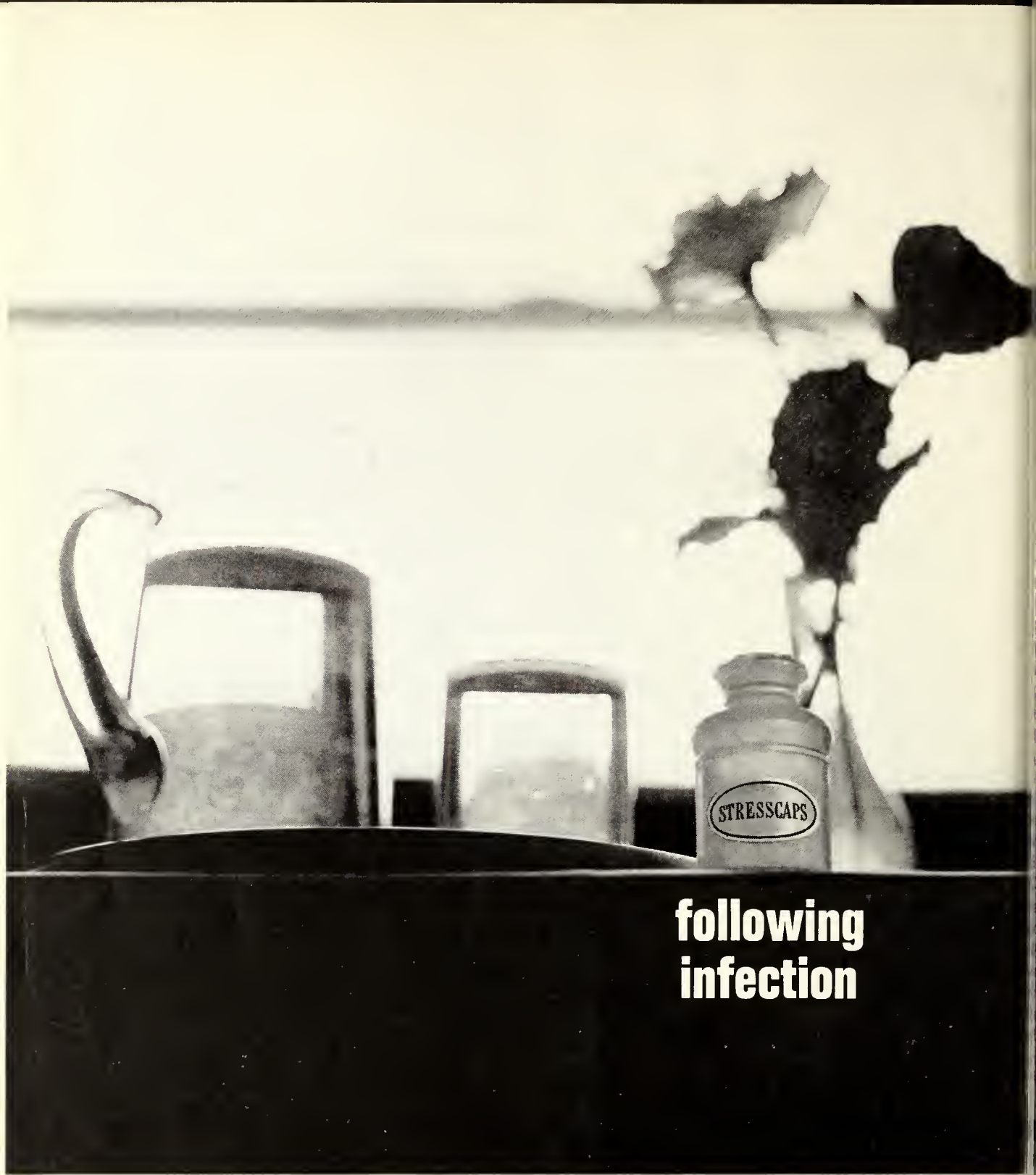
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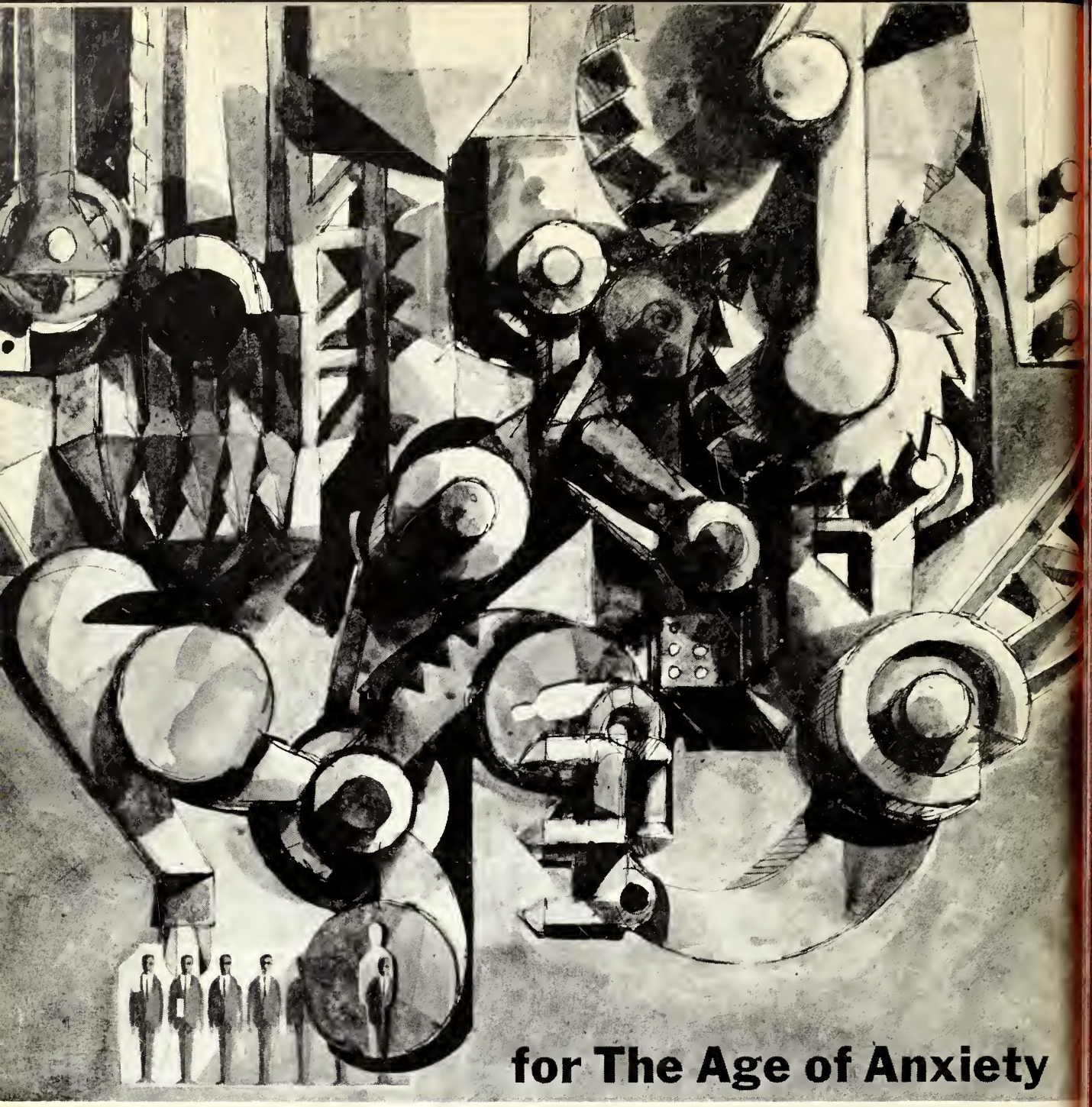


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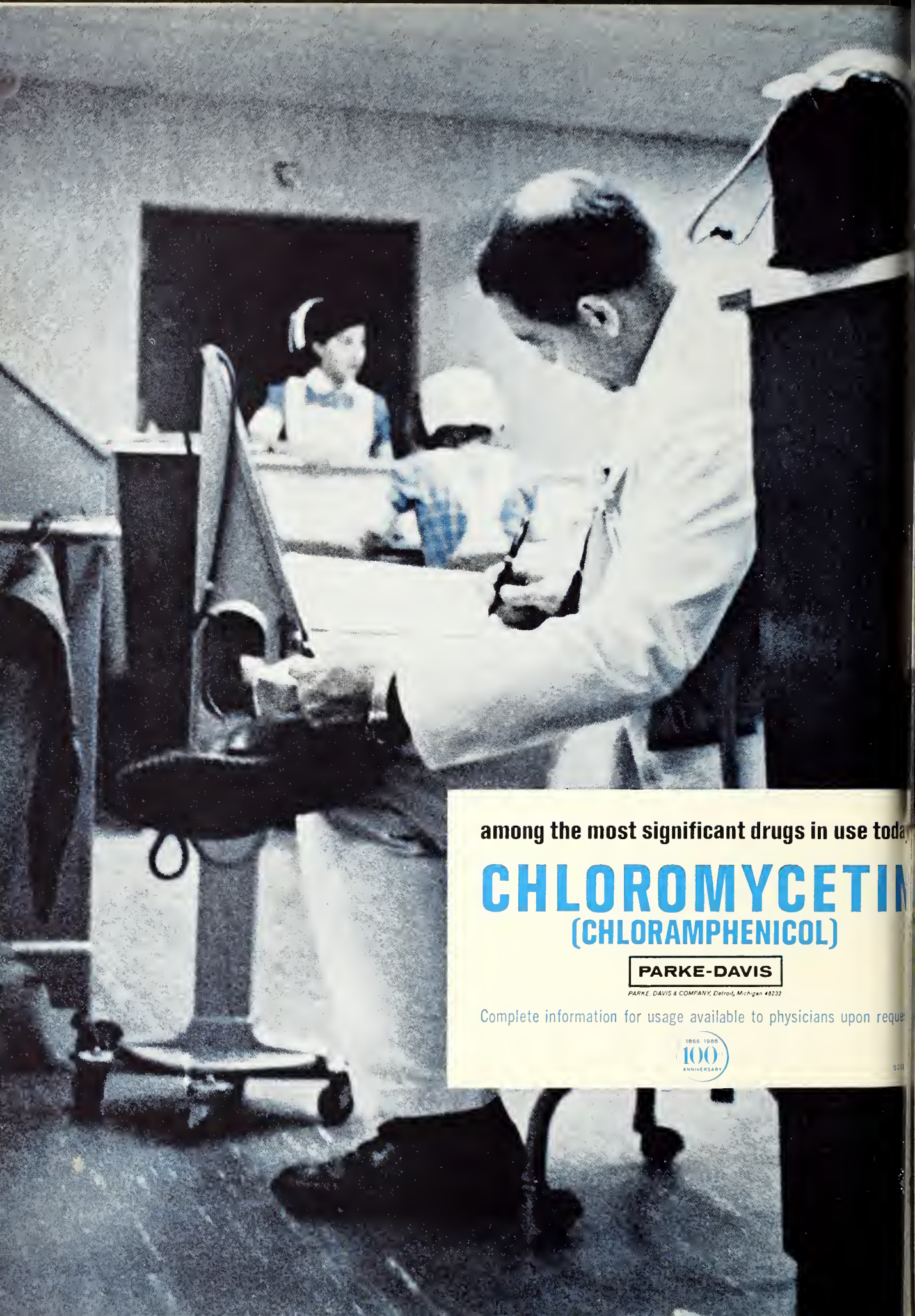
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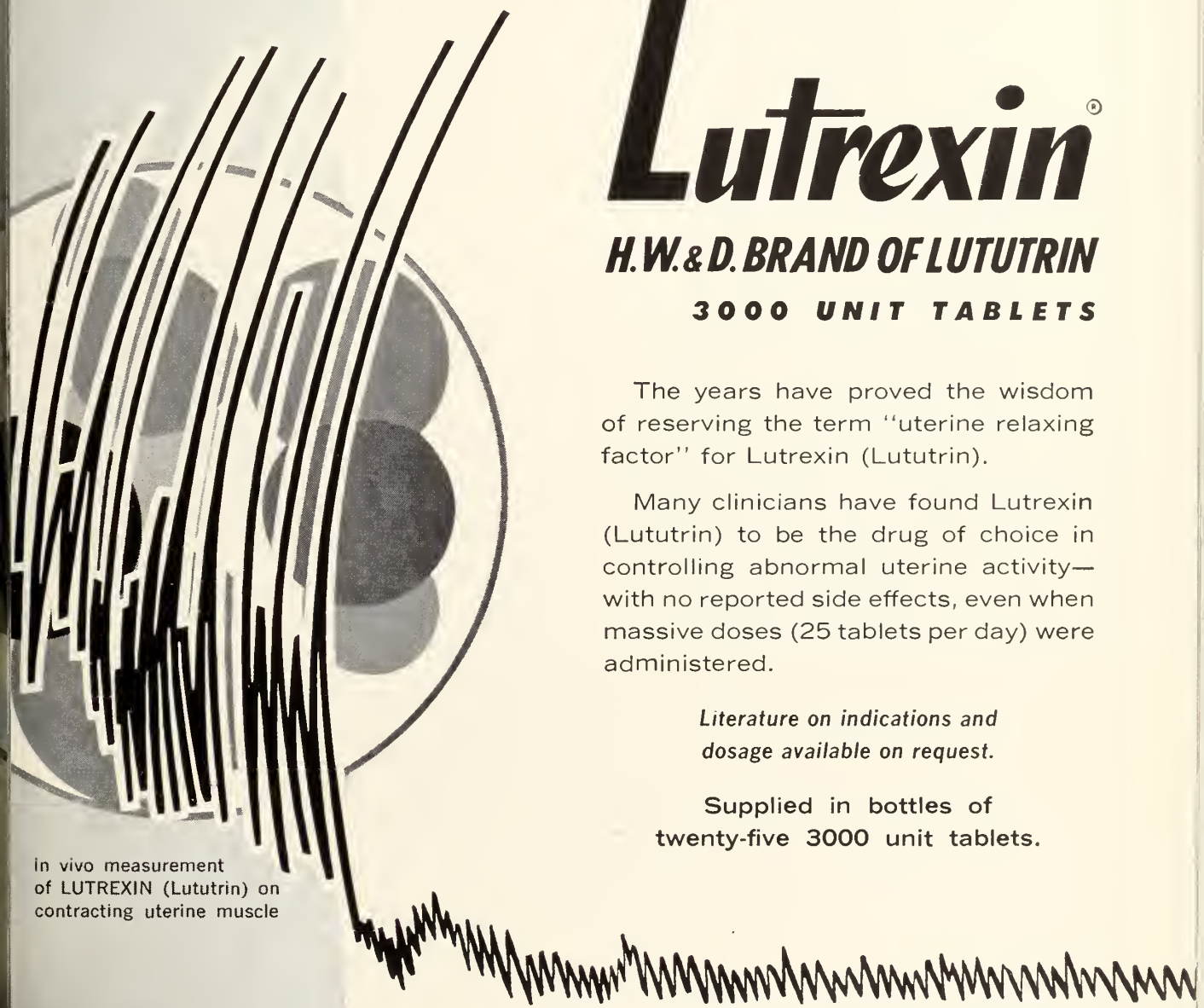
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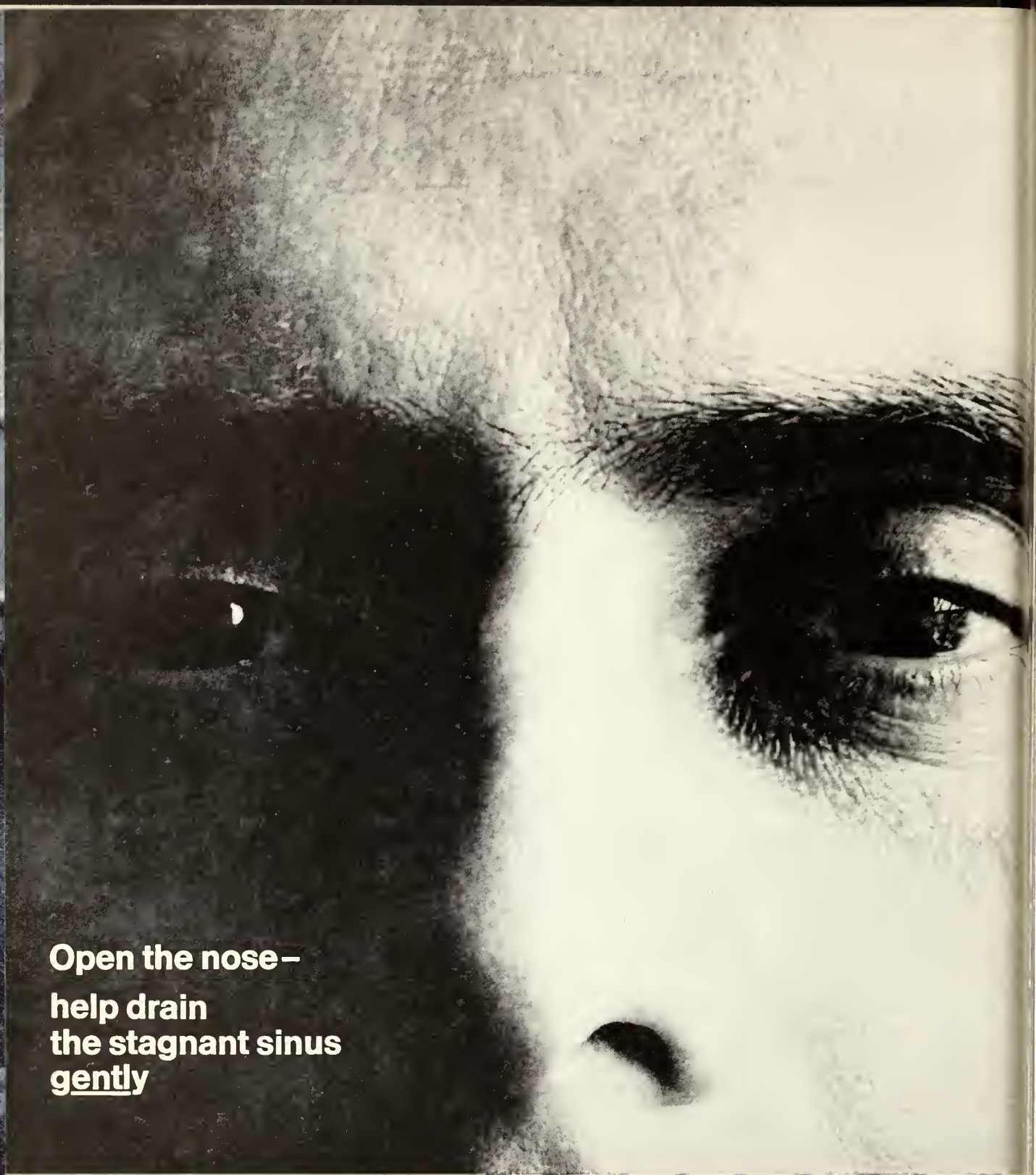


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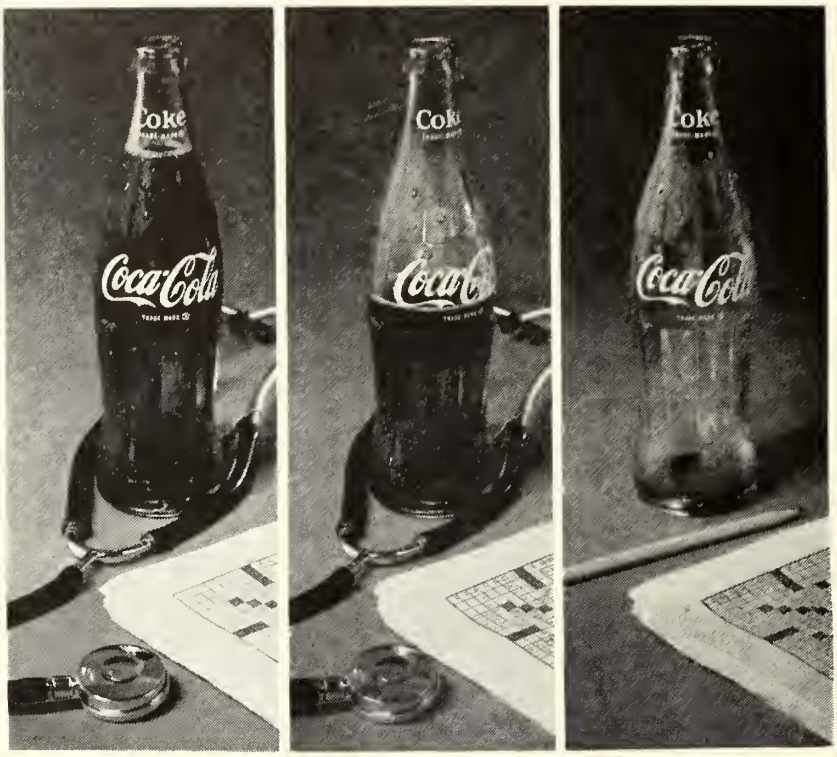
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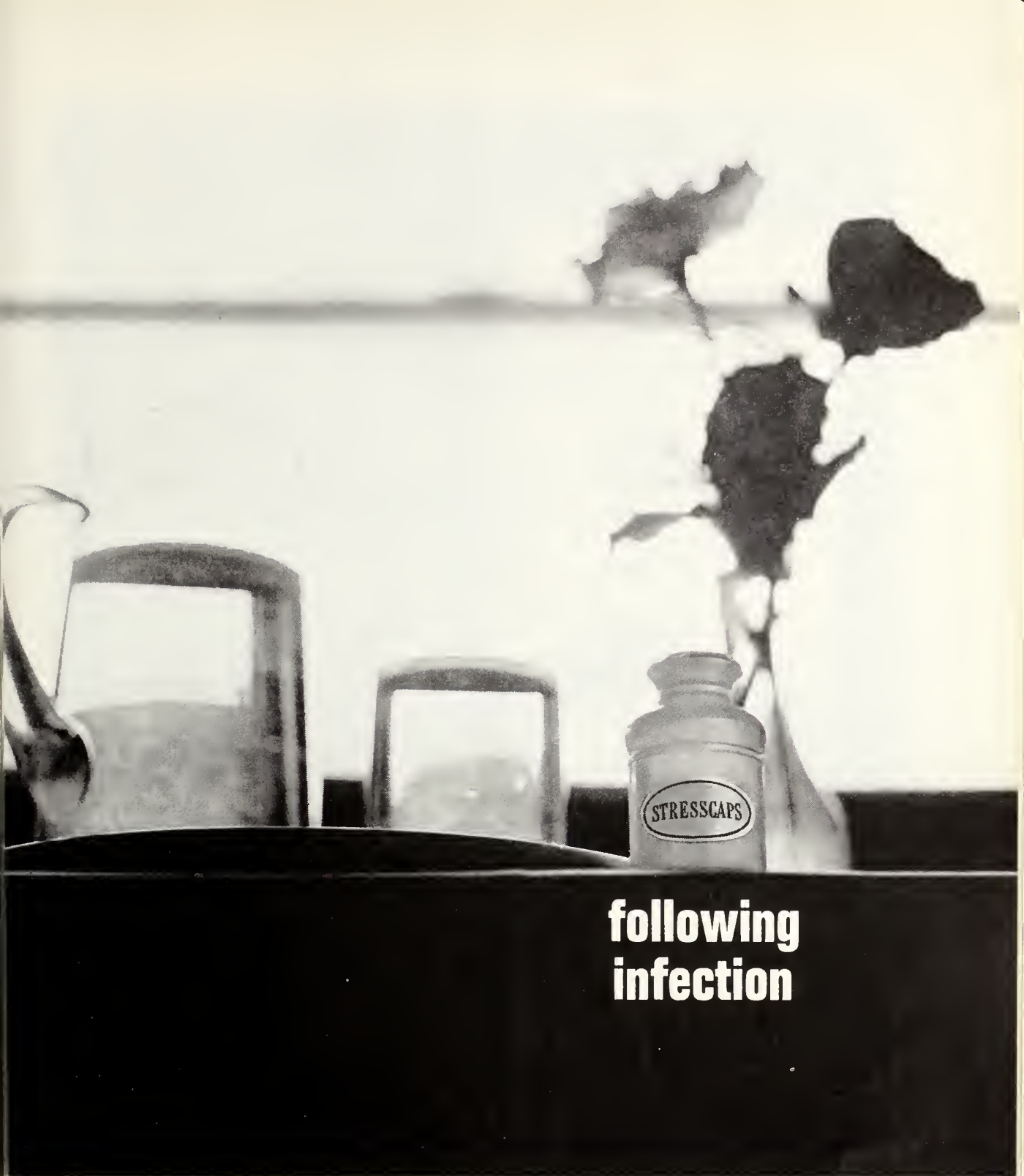
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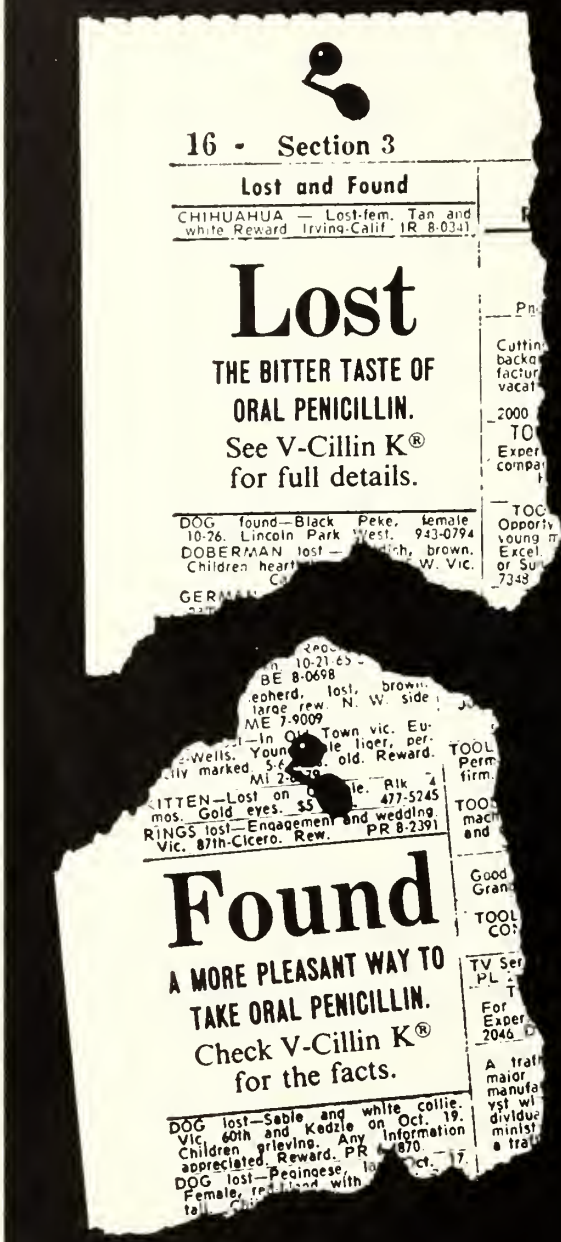
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A Multidisciplined Evaluation Clinic for the Child With a Handicap — A Summary of the First Year's Activities

Edmund N. Ervin, M.D.

In the February, 1964 issue of *The Journal of the Maine Medical Association* we discussed a concept of care for the child with an enduring disability, utilizing the resources of a rural Maine community medical center. Such ideas did not originate with us because multidisciplined evaluation clinics had begun to spring up in scattered teaching centers throughout the country. A multidisciplined evaluation clinic for the preschool mentally retarded child had been in operation here for seven years under the auspices of the Maternal and Child Health Program of the State Department of Health and Welfare. It did remain to determine whether a hospital like the Thayer Hospital would be able to offer a comprehensive evaluation for a child with any type of handicap, what the costs of such services might be, and what might be the implications for clinic services in rural Maine in the future. In July of 1964 the Division of Chronic Disease of the United States Public Health Service, through its Neurological and Sensory Disease Program, approved a project grant to the Thayer Hospital to conduct an outpatient clinic for the child with a neurological and sensory handicap using a multidisciplined team based at the Thayer Hospital, Waterville, Maine, serving six rural counties with a population of 215,000. The primary purpose of the project was service. There were, however, secondary goals to be achieved which are important in the evolution and interpretation of medical care and public health services originating in a regional community hospital in rural Maine. We proposed to accomplish the following specific goals:

1. To provide diagnostic, treatment, and rehabilitative services on an outpatient basis for the child with a handicap; the slow learner, the mentally retarded, the brain injured, the partially sighted, the emotionally disturbed, the psychotic, the epileptic, the physically handicapped, those with speech and hearing problems and the ones handicapped by coexisting chronic illness. Thereby to discover in the child with a handicap, abilities as well as deficiencies, and interpret such facts to parents so that they might invest their energies in areas of greatest potential for development.

2. To offer the child with a handicap, his family, and his physician a continuing program of care, support, and guidance through home visits by a public health nurse and a medical social worker, and speech therapy when needed. Periodic reevaluation by the total clinic team would determine progress and anticipate the changing needs of the child.

3. To demonstrate the public health advantages of a Regional Community Hospital extending its services as a referring and teaching center to the child with a handicap.

a. As a local resource for teaching health personnel how to recognize a child with a handicap and what can be done for him in a clinic, in his own home, and in his community. This educational effort would be aimed at case finding and a demonstration of the best means by which continuous program services could be provided.

b. To determine the cost of a Regional Community Hospital extending its services as a referral and teaching center through a multidisciplined team approach to the child with a handicap.

4. To coordinate patient needs and community resources by analyzing existing resources, developing effective referral patterns, promoting services to fill unmet needs and stimulating improved relationships between hospital, patient, family, physician, service agencies, and the community.

Despite the increasing concern for the welfare of the

handicapped child there is a decreasing utilization of the tremendous amount of knowledge which is accumulating concerning him for many different reasons. The needs and the problems of the child with a handicap are multiple and complicated and highly specialized clinic services have been developed throughout the country to handle them. With all, there has been a tendency to fragment the human body because of these specialized services and we often do only a partial job in the evaluation of the handicapped child because we have lost an understanding of the whole child and the total family situation, as well as the relationship of his handicaps to his total functioning. Secondly, it is a considerable chore for the practicing physician to keep abreast of the flood of new ideas in his own sphere of interest and it is impossible to expect him to be aware of the developments in the various medical and paramedical disciplines which relate to the numerous problems of the child with a handicap. In addition, there has been a serious lack of coordination among the various professional groups and agencies who see these children. How much time and money, both public and private, are consumed with these efforts. There are numerous examples of fragmentation, duplication and delay in the provision of services. There is a need for integration of services into one facility with the use of many disciplines to contribute to comprehensive diagnostic evaluations in order to give the practitioner a proper diagnosis for the individual whom he is treating. Finally, the Congress of the United States in amending the Social Security Act through H.R. 3699 "Child Health and Medical Assistance Act of 1965" has recognized the needs of the multi-handicapped and has prescribed that health care and services for them must be of a 'comprehensive nature'.

Method of Procedure

1. *Clinic Procedure*: Clinic referrals are accepted from physicians, osteopaths, public health nurses, public and private social agencies, and schools from the six county area served by the Thayer Hospital. At the request of federal consultants from the Division of Chronic Disease the program has been enlarged to accept any child with a neurological and sensory handicap who might apply for evaluation. Initially we had accepted children up to the age of twelve years and this has now been expanded through adolescence. Initial contact of clinic personnel with the patient is made jointly whenever practical by the social worker and the public health nurse. They work in conjunction with State workers as necessary. There is a close liaison with the State public health nurses as well as those nurses employed by school department in this area. This initial visit provides an exlent opportunity for a first hand evaluation of the patient and his family in their familiar surroundings, and also provides an opportunity for obtaining a more thorough medical psycho-social history. On this visit the social worker can discuss in a more leisurely manner many of the family's questions in regard to clinic procedures, finances, etc. In addition it provides the clinic staff with an opportunity, if necessary, to meet with the referring physician and/or other agencies or personnel in the community who have a more intimate contact with the family in the past. Reports of the nurse and social worker are then available to the staff prior to the time of the patient's first visit to the clinic. In addition, the social worker has an opportunity to initiate the clinic record and thus eliminate to a large extent the long waiting period on the day of the initial visit. With much of the intake material at hand, as well as other reports which become available to this, it is then possible to arrange for necessary laboratory and consultative services which may be needed. As a result it is possible to stagger appointments with individual team members and make more effective use of clinic services through better planning. Each child receives a complete physical and neurological examination. Consultation is requested from the following specialties as needed: Psychiatry, Psychology, Rehabilitation and Physical Medicine, Speech Therapy, Physical Therapy, Orthopedics, Ophthalmology, Genetics, Nutrition, Otolaryngology, and Reading Psychology. A case conference technique is used to discuss the patient, his family, and community resources. From this composite and comprehensive evaluation plans are formulated for therapy-rehabilitation, home care follow-up, and special education. Following the development of these plans the project director meets with the child's parents to interpret the findings of the group and answer the parents' questions in an attempt to develop a better understanding of present and future problems. Continuity of patient care is extended to the patient's home and community by the public health nurse, the social worker and the speech therapist. The clinic nurse and social worker determine the appropriateness of local and community resources that might be used by the patient and his family.

2. *Education*: As important as the clinic facility is, one of the key figures is the referring physician. He must come to appreciate that children are different, the significance of these variations, and that these may need investigation. This means a considerable educational effort. The calibre of the reports which go out to him will do some of this but the referring physician or a representative from the referring agency is invited to the post-clinic session when the case concerning his patient is discussed. This meeting attempts to create an awareness of the patient's needs and problems, emphasizing the importance of continuing patient care. An effective liaison between the clinic and the patient's physician is an important educational feature for clinic personnel because it will keep the problems of the children constantly exposed. This will make their recommendations more practical and knowledgeable, thus the clinic is a two-way teaching street for the clinic team in the hospital and for the referring physician or service agency in the outside area. Patient and family education is conducted not only at Thayer Hospital in the clinic setting

but also in the patient's own home and community. Community education aimed at coordinating existing resources and stimulating the formation of new services will be conducted. Thayer Hospital is known as a teaching hospital for the scope and number of its educational programs. The oldest formal teaching program–Rehabilitation of the Deaf Child–has grown in fifteen years from a class of three to eighty-five children. In addition the clinic serves as a teaching resource for all health personnel with opportunities for in-service training and observation for all disciplines.

3. *Research*: It was not planned to conduct an intensive research effort in any particular aspect of this clinic service. The focus was to be on service, not research. However, it is intended to conduct some measurements of applied research:

a. Since this is a hospital-based program the opportunity more readily presents itself to correlate whenever possible prenatal influences and their effects on the child with a handicap.

b. Case finding will be a function of all members of the clinic team through physicians, school and social agencies with measurements of the magnitude of the problem by type of disability, source of referral and geographical distribution.

c. It is proposed to determine costs of this multidisciplined hospital-based effort in order that other community hospitals may benefit from this experience. Although this type of effort has been satisfactorily demonstrated elsewhere in hospitals in Maine, this need is for locally applied research to recognize the public health advantages of becoming referral and teaching centers.

4. *Project Evaluation*: 1. Clinic procedure–There have been no significant changes in the clinic procedure as outlined in the original plan but there have been refinements of organization as noted in previous reports. The routine evaluation of the patient was expanded to include vision screening and dental examinations. Certainly the questionnaire, completed by the parents and returned before an appointment is confirmed, has been invaluable in expediting patient flow, arranging consultations and laboratory procedures in advance. It has reduced the duplication and delay in services. It has eliminated eight (8) cases who were not suitable for evaluation by the clinic.

a. Referrals

New cases admitted to service	88
New cases refused after initial screening	8
Revisits	30
Appointments made and not kept	13

b. Source of Referral	*7/ 1/64-3/31/65*	*7/ 1/64-6/30/65*
Physicians and Osteopaths	31	49
Public Health Nurses	4	7
School Nurses	5	10
Child Welfare	1	3
Kennebec Mental Health Clinic	4	5
School Principals	3	12
Social Worker	2	2
	50	88

c. Location of Home

Farm	7
Rural non-farm	29
Small town (500-2,000)	4
Large town (2,000-5,000)	11
Small city (5,000-10,000)	16
City (over 10,000)	21
	88*

*50% of patients come from communities of less than 5,000 population.

d. Economic Status of Family

Fully dependent on public support	3
Partially dependent on public support	8
No public assistance, income $2,000	3
Total income, $2,000-3,999	29
Total income, $4,000-5,999	22
Total income, $6,000-7,999	13
Total income, $8,000 or more	10
	88

e. County	*Age 0-2 years*	*2-6 years*	*6-12 years*	*Total*
Kennebec	1	13	28	42
Somerset	4	9	20	33
Knox	0	0	2	2
Waldo	0	1	2	3
Lincoln	0	0	3	3
Piscataquis	0	0	1	1
Penobscot	0	1	1	1
Sagadahoc	0	1	1	2
	5	25	58	88

f. Consultations

Ophthalmology		8
Neurology		1
Electroencephalography	21	
EEG – audiometry	1	
Otorhinolaryngology		1
PGSR	5	
Audiometry	2	
Cardiology		1
Orthopedics		1
Reading		1
Psychiatry		9

5. *Consultative Services*: Emotional problems often complicate and aggravate an intellectual or physical handicap. At an early stage we set up a reciprocal relationship with the Mental Health Clinic of Kennebec County which has been invaluable. Late in 1964 the services of Dr. Alice Fleming, child psychiatric consultant to this clinic, were made available to us. Dr. Fleming may interview two patients and their families and we may present an additional two or three cases for review and advice. Following this the whole staff meets with Dr. Fleming to discuss exhaustively all the aspects of each case. This has been a tremendously stimulating experience and has helped to give us a greater orientation psychiatrically. We expected that emotional problems would be evident in many of our cases, either from improper guidance and management or from none at all. We realized that they might complicate the solution and disposition of such cases but that we needed the

staff and organization which could understand and consider the emotional aspects of the child with a handicap and be able to offer reasonable and appropriate therapy. Our arrangement with Dr. Fleming has been an excellent one but this is an insufficient effort and other services at a different level of competence and training are needed. One fourth of our present case load has had an emotional problem as a major feature; consequently we are hoping to add an individual in clinical psychology with a strong background of experience with young children who has competence in projective techniques, as well as routine psychometric testing, in addition to the background of supervised training in child psychotherapy.

6. *Continuity of Patient Care*: Problems in social adjustment necessitating follow-up services after the diagnostic workup have made some type of case work service advisable. Such services are now on a regular planned and continuing basis. These services help with problems in parent-child relationships, with family-school relationships, as well as other case work services dealing with financial problems and medical care.

In the cases where the parent-child relationship is thought to be the basic problem case work goals have been to help the parent(s) express their feelings, to gain insight as to how these feelings have been operating in their management of the presenting problem, and to become more sensitive to the child's feelings. When the major problem has been in the family relationship with the school case work services have been directed toward helping the parent and teacher understand the child's potential for academic accomplishment, what the child's social environment is, how these factors relate to his emotional problems, an interpretation of clinic recommendations and how they can be implemented. Medical care as recommended is discussed with the family for (1) assessment of their understanding of the patient's condition and the prescribed treatment; (2) clarification of areas of misunderstanding with patient. Continued work with the family has as an ultimate goal the modification of some attitudes and the alleviation of some fears that will free the family to make full use of medical facilities. Practical help is given in making resources known to them and in mobilizing and coordinating existing community resources. For example when the family is making–or can make use, of more than one medical or social agency, functions and roles must be clearly understood that real service is given and coordinated rather than in a fragmented way.

The public health nurse is involved in the previously mentioned effort, in addition makes other contributions. Her counseling is directed toward a better understanding of the problems and potentialties by a consideration and discussion with the parent of (1) child rearing practices, (2) health protection, (3) the needs of a growing life. She offers emotional support and reassurance, also makes recommendations which can be translated into patterns of life. Our public health nurse has been interested in developing community resources which may be of use in handling some of these problems locally. This means (1) the augmentation of present services and the development of new ones, (2) effective interagency referrals, and (3) improvement of communications between the hospital and the local school and social agencies.

It is too early to make any definitive comments about our speech program for our consultant has only been available to us for the past three months of the first year of the program. However, there has been no speech pathologist or therapist in this area and at the present time he is being deluged with requests for consultation and therapeutic efforts.

Follow-up services have been offered to patients and their families in various ways in an effort to provide continuity of care. Many of these are diagnostic efforts which are also supportive by reason of eliminating previous diagnostic errors and misconceptions. It has been necessary to see only fourteen parents again to discuss final recommendations or to clarify previous ones.

It is interesting that we have been able to refer only four (4) patients back to their physicians. This is explained by the absence of available case work services in the local communities as well as other resources such as speech therapy, etc. It may be that our hope of developing such community-based facilities is unrealistic (see cases listed under Project Evaluation) and that a community hospital such as Thayer must be the basis of such patient care.

7. *Case Material*:

Chronic Brain Syndrome (abnormal EEG) *	5
Orthopedic	4
Epilepsy	13
Vision	6
Hearing	2
Cleft Palate	1
Emotional	23
Speech	24
Mental Retardation	39
Heart	4
Cerebral Palsy	6
Skin	1
Congenital Hydrocephalus	1
Reading	3
No Disease	5
Total children 88	137 diagnoses

*Definition – This diagnostic term has been used because of its inclusion in coding classifications. It refers to the neurologically handicapped or brain damaged child as defined by Ingram "A condition of cerebral abnormality which, though not sufficiently severe to cause easily identifiable symptoms of cerebral palsy, is severe enough to cause minor motor dysfunctions, epilepsy, learning difficulties or abnormality of behavior."

It is impossible with the amount of case material which we have at hand to assess the value and effectiveness of this effort in a meaningful way after this initial year of the project. There are suggestive trends without conclusions.

a. The increasing demand for service is an indication of need (see source of referral).

b. Fifty percent (50%) of patients come from communities of less than 5,000 population. Our hope of using or developing community-based services may be unrealistic because the type of continuing services which are needed can only be offered in a regional medical center. However, through the invitations to attend the conference sessions and the reports which are sent to them, we are creating an awareness of this effort among physicians and school authorities.

c. It is interesting that with the clinical and laboratory facilities of this hospital, the staff of this clinic has been able to offer a complete and comprehensive evaluation in every case but three.

Referred to other hospitals:	
Orthopedic and Neurology (Mass. General Hospital)	1
Surgery (Boston Floating Hospital)	1
Cardiology (Maine Medical Center)	1

d. We have said that these children are multi-handicapped. This is shown in the summary of diagnoses (of significant handicaps only) of our case material.

e. The small number of significant orthopedic handicaps and the incidence of emotional problems was unexpected.

f. The demand for speech therapy is noteworthy.

g. No attempt to assess cost will be done until completion of the project.

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Why Prophylaxis for Ophthalmia Neonatorum?

Richard H. Dennis, M.D.

It would seem that prophylactic care of eyes of the new born is so well accepted that it needs no discussion. And indeed it is, in concept. Yet it is surprising to find the divergence of opinions on the method of carrying it out. The rapid on-rush of constantly newer antibiotics has many thoughtful doctors wondering about the wisdom of standing pat on older methods. Through the years, research has been instituted, surveys conducted and series have been run on many antibiotics and techniques, which have nothing in common with the old Crede' method except that they are carried out on babies. Some states, in fact, have curtailed the latter method, and allow other, more modern ways. (N. Y. City, Jan. 1955). In some parts of England, largely as a result of work done by Ida Mann, Streptomycin has been used. In European circles, a good deal of interesting work has been done using "Desogen" a quarternary ammonium base and good results were reported.

Perhaps as a result of the current disturbing upsurge in gonnorrhea in the general population, or perhaps due to a general confusion as to what prophylaxis is at present most widely accepted, some of the ophthalmologists were approached for advice as to the current practices.

They referred the request to the Maine Medical Association's Eye Section. The subject was then included on the agenda for discussion at the last meeting. At the conclusion of the meeting, it was suggested that the results be reported through the Medical Journal.

There are two diseases of the conjunctiva of the new born which must be differentiated; ophthalmia-neonatorum and inclusion blennorrhea.

The first is caused by gonococci contacted while passing through the birth canal of infected mothers. The infant's conjunctival sacs are thus heavily infected. A diffuse copious purulent discharge develops within two days. If the condition develops later than 48 hours, the infection was not picked up at birth, or is non gonorrheal. The heavy, purulent discharge is almost unique, but etiologic diagnosis can be made by finding gonococci in the discharge or in epithelial scrapings.

A severe idiosyncratic reaction to the silver nitrate which is given at birth in the Crede' method may be confused with gonorrheal ophthalmia. However, it usually disappears in three days without treatment and of course, no gonococci are found.

The second condition associated with the newborn is inclusion blennorrhea. This is a moderately severe infection of the conjunctival sac. However, it occurs in 5 to 7 days, is self-limited, and will disappear by 10 to 14 days. It may be diagnosed by epithelial scrapings, wherein inclusion bodies are found. Although self-limited, its disappearance may be hastened by irrigation and broad spectrum antibiotics.

In 1880-81, Crede' surveyed a large number of births in a "general hospital" and found a 10% incidence of gonorrhea neonatorum. After considerable investigation, he finally concluded that the best prophylaxis seemed to be the instillation of 2% silver nitrate in the eyes of new born babies, followed in 30 seconds by irrigation with saline. He reported a series of 200 consecutive cases so treated without a single infection, an unheard of event in that time. It was later found that 1% solution was just as effective and did not require irrigation with saline. From that time, until the inception of the antibiotic era, the Crede' method was found to be safe, easy to use and effective. It was widely employed and indeed was required by law in many states and countries. (N. Y. 1922)

When antibiotics arrived, they were, of course, investigated as possible prophylaxis for ophthalmia neonatorum. In the 1950's, there were reports from many responsible investigators. A few of these follow.

Ida Mann[1] in 1954 reported an extensive series. In it she used several modes of approach.

1. Simple saline irrigations.
2. Silver nitrate.
3. Streptomycin.
4. Penicillin.
5. Sulfonamides.

She concluded that Streptomycin was the most effective when fresh solutions were used. However, she found that silver nitrate was very nearly as good, was more stable, and was usually more easily obtained. She found that Penicillin was slightly less useful than Streptomycin and sulfonamides least effective. However, she felt that the best prophylaxis of all was adequate prenatal and natal care with delivery carried out in a modern, well equipped hospital.

H. L. Ormsby[2] in 1955 reported a series of 8418 births in which he investigated!

1. Silver nitrate.
2. Sulfacetamide.
3. Sulfathiazole.
4. Sulfadiazine.
5. Penicillin.

He had a control series with no prophylaxis which showed a relatively high incidence of gonorrhea. He reported that the Crede' method seemed to be the most practical. Even though the antibiotics were effective in varying degrees, they were unstable and engendered sensitivities. He also reported that the agents had no direct effect on inclusion blennorrhea.

Schoenenberger, F.[3] reported in 1957 on the use of "Desogen" - a quarternary ammonium compound like Zephiran. He felt that it was an effective, nonirritating conjunctival bacteriostatic agent. He reported 310 cases and concluded that this substance was effective and stable. He advocated instillation of a .05% solution to remain in the conjunctival sac for five minutes.

Salvesberg, H.[4] in the same year, made a survey of 34 European Hospitals and came to the following conclusions!

1. Some prophylaxis should be used.
2. "Desogen" seemed to be the best.
3. Silver nitrate or Silver acetate was nearly as effective.

Margeleth,[5] in 1957, reported on 7,774 infants. He divided them into two series. In one he used Penicillin ointment as a prophylactic. In the other, he used Bacitracin ointment. He found no severe reaction to either. He concluded that, of the two, Bacitracin was more effective and had minimal initiation. However, he stated that he thought the best treatment was simply observation.

From 1956 to 1958, Greenberg and Vandow[6] collected a series of 472,000 babies from General Hospital populations and from smaller "propietary" hospitals in New York City. They found 49 cases of gonorrheal ophthalmia neonatorum (.0017). They investigated the following methods of prophylaxis.

1. No prophylaxis.
2. Silver nitrate.
3. Bacitracin ointment.
4. Intra-muscular Penicillin (33 000) babies using 50,000 units.

They concluded that prophylaxis should be used and that Penicillin was the most effective and advised its use. They had no cases at all in babies so treated.

Dr. Thygeson, in his section of the Lancaster Course for 1964, discussed gonorrheal ophthalmia neonatorum. He advised, as the most effective agent, Penicillin ointment, 100,000 units per gram. He felt that the Crede' method with 1% silver nitrate was nearly as good.

In Maine, at the present time, the Crede' method is suggested by Dr. A. Ashley's department. She, however, reports only 1 case in the last five years.[7]

The Eye Section of the Maine Medical Association, after considerable discussion, agreed that prophylaxis was necessary and the Crede' method was still probably the most suitable for the following reasons.

1. Effective.
2. Safe in the proper strength.
3. Stable in the customary method of packaging.
4. Non-sensitizing.
5. Easy to use.

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The Natural History of Disease

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Another physician and I had decided to report for this issue an interesting case of a lady with cancer of the breast. The point to be emphasized was that, although she had had the disease for ten years, had fractured her hip, and had recently developed bone metastases, she made a good enough recovery with scientific plus humanitarian treatment, to return home. We were sure that this recovery was quite unusual and would emphasize the value of aggressive therapy even in advanced stages of the disease.

We were sure until we looked deeper into the problem. Then we found that we really had no knowledge of the natural history of this disease. We had felt that her course following treatment had been better than normally could be expected. However, in discussion with several specialists on our Tumor Board I found that they had seen several similar cases. Next we looked into the literature. The State of California Department of Health has compiled statistics on cancer survival.[1]

For cases aged 55-64, whose cancer was localized at time of first diagnosis (the characteristics of the case we were going to report) there is a relative survival rate of 98% for 1 year, 91% for 3 years, 83% for 5 years, 63% for 10 years, and 52% for 15 years! Relative survival rates, "are adjusted for deaths from causes other than cancer which would be expected among persons of the same age in the general population."[2] From these figures it would seem our patient had a slightly better than 50/50 chance of living another five years!

The same publication has an interesting statement in its chapter on "First Course of Treatment."[3] "Survival rates are a measure of the success of therapy in prolonging life, but *because of lack of knowledge of the natural history of disease,* it is difficult to determine the extent to which treatment actually adds to the length of life of the patient with cancer."

This led me to pursue the study of the natural history of disease. It is indeed surprising to find how little our knowledge has advanced in this field. We do have a good understanding of acute and curable diseases. However, knowledge of the benefits of various treatments given for symptoms, or given for chronic diseases and impairments is very shaky. In brief, our ability to diagnose an illness has improved, our ability to choose the proper specific therapy has improved, but our ability to make a prognosis of life expectancy or even functional capability in the individual case has lagged behind.

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Three examples may help to emphasize this point:

1. Lack of knowledge of the true meaning of symptoms:

Gastrointestinal symptoms are still one of the most frequent reasons why adults visit doctors' offices. Yet gastrointestinal disease is not included in the first ten causes of mortality in the U. S. A. How can this paradox be explained? One explanation might be that most of the symptoms were cured by treatment. Another might be that they were the first clues of a developing disease of another organ or system. If this latter explanation is true–or even part true–many cases of early and possibly reversible disease are being missed every day. The only way of learning about the natural history of gastrointestinal symptoms is for practicing physicians to do long-term follow-up studies of these patients.

2. Lack of knowledge of the prognosis of a chronic disease in the individual patient:

Early in the course of nearly any chronic disease, it is almost impossible to judge the individual's ability to resist or adapt to it. Certain factors such as age, sex, associated disease, heredity, and living pattern, are known to be important. However, of all cases in semicoma one day after a stroke, how many will go on to full functional recovery, how many will hobble through life, how many will have a recurrence, and how many will remain in coma or die? This we are unable to predict as yet. The same is true of peptic ulcer myocardial infarction, arthritis, or bronchitis. Yet the one thing the family and the patient wants to know is prognosis. There are now fairly accurate statistics on what percentage will have what result. There is still inadequate knowledge about which percentage group your patient will join. Here again, follow-up studies by practicing physicians who know the patients intimately seem the only answer.

3. Lack of knowledge of health:

We are supposedly leaders in the health profession. Opportunities to study health rather than disease occur daily. It is well-known that given any stress–be it a tubercle bacillus, a fall, an emotional crisis, or cigarette smoking–various individuals will react in various ways: full-blown disease, subclinical disease or no disease. Why some are susceptible and some are resistant is vital information. Because we don't have good studies of health, we tend to equate certain diseases to certain stresses even though we know the same stress may cause no disease or a completely different disease in another individual. We know it, but don't dare say it. Again this gap in knowledge can only be filled by studies done by doctors in practice.

The Views of Sir James Mackenzie

I first became interested in studies of the natural history of disease when I read *The Future of Medicine* written by Sir James Mackenzie in 1919.[4] Here are some quotations from this rather timeless book–

"The manner in which the investigation of symptoms should be pursued has not been understood, and it may be taken for granted that the progress of medicine will be greatly hampered until the study of symptoms has been thoroughly prosecuted. . . From this consideration another principle appeared, that the individuals, conducting an investigation, should have had the opportunity of seeing disease in all its phases, and of observing the symptoms it produces. . . It is manifest that neither the laboratory worker, nor the hospital physician, who are the persons mainly concerned with research, have this opportunity. . . It is evident that only one class of individual has the opportunity for acquiring this knowledge, and he is the practitioner."

"Medicine includes within its compass a number of branches which are called sciences, as physiology and bacteriology, but none will be found better to deserve the name of science than that of prognosis when its significance is fully realized. . . This is a kind of knowledge that is essential in the practice of medicine and which no academic investigator can ever acquire."

"We need not be surprised if the public looks with trust on the magnificent temples dedicated to research and ignores the simple methods of the practitioner with whom lies the future of medicine."

"In medical schools there ought to be a teacher, or several teachers, whose experiences enable them to take a wide outlook on the whole field of disease. The man who should take this position is an experienced practitioner, and he ought to be the dominant figure in medicine. . . At one time the physician did occupy a dominant position, but during the last fifty years the laboratory workers, and the surgeons, have made such progress in their special departments that they usurp the physicians position. To a great extent the physician is himself to blame. He has allowed himself to be so dazzled by the inventions of his colleagues, and has accepted their methods and ideas, not recognizing the limited scope of all such methods, while his own vastly more important field has been neglected."

Discussion

The hopes in this book have not come true forty-six years later. The future of medicine has not turned out to be based on the teaching and research of practitioners. It is because of this that we are top-heavy in complex knowledge of isolated facts about certain diseases, but have advanced little in our day-to-day handling of symptoms and the patient manifesting them.

A more modern book that states the same conclusions is *The Natural History of Disease* written by John A. Ryle.[5] Although he was a professor at the Oxford University Medical School, he agreed with Mackenzie on the need for research and teaching done by practicing physicians if the knowledge of the natural history of disease was to progress.

Knowledge of the natural history of disease is vital if we are to have a true measurement of the meaning of symptoms, individual variation in health or disease, and results of treatment. It is a body of knowledge that can serve to integrate and simplify medicine in this present thrust towards complexity and specialism.

Yet progress in the understanding of the natural history of disease is lagging. The principle reason is that, by the very nature of its content, it must rely on observations made by practitioners on individual patients over long periods of time.

Practitioners are preoccupied with service, and think of research as something done by academicians in laboratories. This brief review is done to emphasize that knowledge of the natural history of disease can advance only if practicing physicians add observational research to their presently crowded days.

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PUBLIC LAW 89-74

Public Law 89-74 went into effect on February 1, 1966 to curb drug abuse. It affects you as follows:

- The law establishes special controls over depressant and stimulant drugs.
- Written prescriptions must include the patient's name and address as well as the date.
- Prescriptions for such drugs will be refillable for only five times or for six months, whichever is less.
- Records must be kept of manufacture, sale, *receipt* and *distribution* of such drugs.

The physician must keep records under Public Law 89-74; however, *they are not required to keep records as a consequence of this law unless, in the course of their practice, they dispense the drugs referred to and charge for them.*

> "The provisions of paragraphs (1, Records) and (2, Inspection) of this subsection shall not apply to a licensed practitioner . . . with respect to any depressant or stimulant drug received, prepared, processed, administered, or dispensed by him in the course of his professional practice, unless such practitioner *regularly engaged* in *dispensing* any such drug or drugs to his patients for which they are *charged*, either *separately or together with charges for other professional services.*"

Nonsuppurative Otitis Media

LORING W. PRATT, M.D.*

Nonsuppurative otitis media is a disease which has become more frequently diagnosed in recent years than in the preantibiotic era. The terminology is varied. Its synonyms are catarrhal otitis media, secretory otitis media, and eustachian catarrh, and it results from obstruction to the normal ventilation of the middle ear via the eustachian tube. The reasons for obstruction of the eustachian tube are many and varied, but any chronic condition which interferes with adequate ventilation of the middle ear (Fig. 1) may produce this disease.

The incidence of nonsuppurative otitis media has been found by Armstrong[1] to be higher in children of less than eight years. Seventy-eight percent of a series of one thousand and sixteen cases, treated by him, were found to be in this group. Nonsuppurative otitis media has been diagnosed more frequently in recent years, and this increase is due to two unrelated factors.[2] In the first place, a certain amount of apparent increase is due to improved understanding and awareness of the problem and to earlier and more adequate treatment for it. The second factor, which accounts for a real increase, is the widespread use of antibiotics. In many instances, suppurative otitis media is apparently successfully treated by means of antibiotic therapy, but a residual sterile effusion often remains in the middle ear as the result of an unresolved eustachian tube obstruction. Thereby, nonsuppurative otitis media is left to plague the patient. This is particularly found to be the case in small children who are not able to complain about stuffiness and deafness as readily as adults. In young school children, deafness noted by the teacher, or by routine test, may be the first sign of trouble.

The etiology of eustachian tube obstruction is varied. It may be an obstruction from an acute upper respiratory infection, an allergic upset, or an acute episode of barotrauma, or it may be a chronic obstruction from a chronic upper respiratory infection, hyperplastic adenoid, allergic disease, sinusitis, or malignancy of the nasopharynx. These comprise the most common causes of eustachian tube obstruction which are related to nonsuppurative otitis media.

Nonsuppurative otitis media may be either acute or chronic. In the acute form, such as is seen in aerotitis media or otitic barotrauma, there is an acute effusion in the middle ear due to sudden change of the pressure relationship between the ambient atmospheric pressure and the pressure within the middle ear. (Fig. 2). It may also occur secondary to acute otitis media, in which the bacterial part of the infection is sterilized by the use of antibiotics and a sterile catarrhal effusion persists, because the obstruction of the eustachian tube is not also simultaneously cleared up.

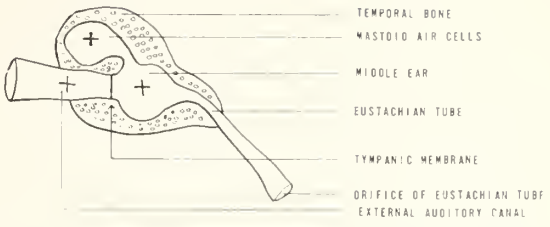


Fig. 1. Shows normal ear – external canal, tympanic membrane, middle ear, mastoid cavity and eustachian tube lined with thin mucous membrane. The + signs indicate normal atmospheric pressure in the external auditory canal, the middle ear cavity and the mastoid air cells.

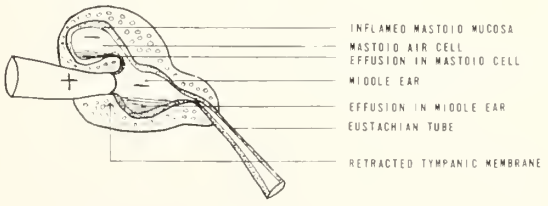


Fig. 2. Shows nonsuppurative otitis media in a well developed form. The air pressure in the middle ear and mastoid cavity is reduced (—) as compared to the normal pressure (+) in the external auditory canal. Fluid effusion is seen in both middle ear and mastoid cavities. The inflamed mucosa of the eustachian tube is clearly seen, especially where it is constricted as it passes through the narrow part of its course in the temporal bone.

Chronic nonsuppurative otitis media has precisely the same fundamental etiology as acute aerotitis media. The latter arises on descent in an airplane (or on descent in diving) when the air which escaped from the middle ear via the eustachian tube on ascent is not permitted return passage through the eustachian tube into the middle ear, leaving a differential in pressure between the middle ear and the external ambient atmosphere. The partial vacuum thus produced results in an effusion of serum, and sometimes concomitant rupture of small blood vessels, giving rise to hemorrhage into the middle ear. Effusion of this type is usually clear, yellow and watery and is easily removed by aspiration through a paracentesis.

In chronic disease, the pressure differential results from absorption of gasses from the middle ear without replacement through the eustachian tube because of impaired eustachian tube ventilation. Blood circulating through the middle ear mucosa dissolves gasses from the middle ear and reduces the pressure, with a slowly developing, long persisting partial vacuum, which re-

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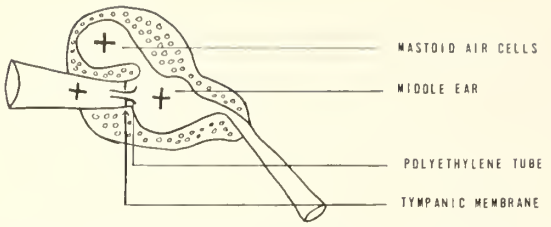


Fig. 3. Shows a polyethylene tube in place in the perforated tympanic membrane equalizing the pressure between the external auditory canal, middle ear and mastoid cavity. The fluid effusion has cleared, the tympanic membrane has returned to normal, and hearing is again normal.

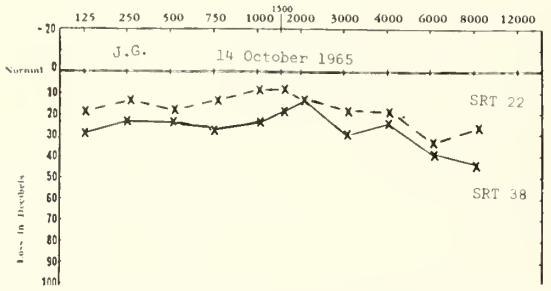


Fig. 4. Shows conductive hearing impairment in the left ear which disappeared, leaving normal hearing five minutes after paracentesis with topical anesthesia and aspiration of fluid from middle ear as an office procedure. The speech reception threshold improved from thirty-eight to twenty-two decibels. There was also complete relief of stuffiness and subjective hearing impairment.

sults in effusion of fluids into the middle ear. In long standing problems the effusion is usually thick, mucoid material which is sticky and stringy and difficult to remove by aspiration through a paracentesis.

The diagnosis of nonsuppurative otitis media is straightforward. The symptoms are characteristic and reasonably constant. The patient usually complains of some hearing impairment, a sensation of pressure in the middle ear, and/or a sensation that there is water in the ear. Usually there is stuffiness, but rarely earache. Sometimes the patient complains that the homolateral side of his head, face and pinna feel numb. Conversation is heard as if the patient had his head in a barrel, so that it seems to be an echo or reverberation. Sometimes hearing acuity changes with change of position of the head.

The physical findings vary slightly with the particular type of nonsuppurative otitis media. In acute aerotitis the tympanic membrane is reddened, retracted and the posterior malleolar artery is injected. The peripheral vessels of the tympanic membrane are also injected. The middle ear is congested, its contents are distinctly yellowish or amber, and sometimes hemorrhage is noted in either the tympanic membrane or in the middle ear.

Acute nonsuppurative otitis media following a resolving acute otitis media purulent usually demonstrates a normal or slightly pink tympanic membrane which is

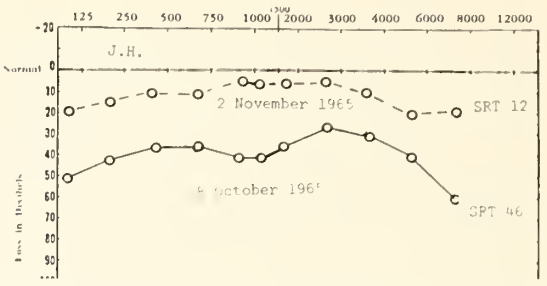


Fig. 5. The lower curve shows hearing at the forty decibel level eleven days prior to paracentesis, aspiration of thick mucoid discharge from the right middle ear, insertion of polyethylene tubing, and secondary adenoidectomy. Hearing seven days postoperatively was entirely normal as seen in the upper audiometric curve.

slightly retracted. The middle ear is congested, has a yellowish tint, or amber fluid may be seen within it. Bubbles are sometimes present.

In chronic nonsuppurative otitis media the tympanic membrane is retracted, slightly pink and congested. The middle ear is congested and amber or dark colored, occasionally having a distinctly bluish color. Sometimes fluid levels and bubbles may be seen within the cavity.

Treatment of this disease is two-fold. The first aim of therapy is relief of the acute symptom, and the second is treatment of the underlying disease. Relief of the acute symptom may be accomplished by opening the tympanic membrane and aspirating fluid which is often inspissated and mucoid (giving rise to the term "glue ear") from the middle ear. If the eustachian tube obstruction cannot be readily relieved in the immediate or near future, by vasoconstriction or by antihistamines, the myringotomy opening in the ear drum may be prevented from healing by the insertion of a six millimeter piece of number ninety polyethylene tubing, with a flared end, into the middle ear through the paracentesis opening, to keep the incision open, so that ventilation may take place through the polyethylene tubing instead of through the usual route via the eustachian tube. The flared end prevents the tube from falling out and the length prevents its loss into the middle ear.

Treatment of the underlying disease is possible only after establishing its nature, and may consist of adenoidectomy, secondary adenoidectomy, antiallergic therapy, treatment of sinus infections, and removal treatment of tumors of the nasopharynx. The lesion is probably most commonly due to either a hyperplastic or a recurrent adenoid. This is readily determined by examination of the nasopharynx, by electric nasopharyngoscopy, by use of the postnasal mirror or by palpation of the nasopharynx. Removal of hyperplastic adenoid or recurrent adenoid tissue by surgery, or nasopharyngeal radium irradation by the method of Crowe[3], is essential to recovery in these cases. Control of allergy by the use of antihistamines or desensitization studies is an important part of therapy. Identification and treatment of a complicating sinus infection is helpful.

Rarely surgical oblation of the mastoid is a necessary part of the treatment. This occurs only when irreversible changes have taken place in the mucosa which favor the continued production of serous exudation in the mastoid and middle ear. The results of this form of treatment are highly satisfactory. In the acute form of the disease, paracentesis and aspiration of the middle ear relieves the stuffiness, tinnitis and paresthesia at once and improves the hearing significantly (Fig. 4). In the chronic form, the paracentesis and insertion of polyethylene tubing results in return to normal hearing in a few days (Fig. 5). When the underlying pathologic process is adequately controlled, removal of the polyethylene tubing leaves the patient with normal hearing. The tympanic membrane perforation heals promptly and spontaneously after removal of the polyethylene tubing in almost all cases.

Conclusions

1. Nonsuppurative otitis media is a common cause of hearing impairment, especially in children and particularly in small children.

2. Nonsuppurative otitis media is the result of eustachian tube obstruction, either acute or chronic.

3. Nonsuppurative otitis media is more common today than in preantibiotic days.

4. Treatment of nonsuppurative otitis media is accomplished by paracentesis, aspiration of middle ear contents, and restoration of nasopharynx to its normal status.

5. Use of polyethylene tubing to maintain a vent in the tympanic membrane is often of temporary benefit in the treatment of nonsuppurative otitis media.

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Bacterial Endocarditis

Paul H. Pfeiffer, M.D.

This case of bacterial endocarditis is presented for several reasons. At our hospital this appears to be a rare condition. Our pathologist claims there has not been a case treated here for the last 15 years. Whether some of the rarity is due to ineptness on the part of the clinicians is open to question. Secondly, this case presented a few unusual features. Finally, it offers an excuse for reviewing the literature of this interesting disease.

Case Report

C. F. is a 15-year-old girl who entered the Thayer Hospital for the first time on the 14th of August, 1965. Two weeks prior to admission she had returned from a camping trip. She had been bitten by black flies while camping and had returned home feverish and with generalized malaise. Due to persistence of the fever, Penicillin was administered a week before she came to the hospital by her physician, but she vomited the oral medication. Three days prior to admission she was still feverish and was noted to have weakness and poor coordination of the left hand. The penmanship of this left-handed girl deteriorated markedly. She had received a full course of Salk vaccine injections 3 years ago. There was no history of rheumatic fever, strep throat, or any surgical or dental procedures.

Her temperature was 104.5°. She appeared drowsy and complained of a slight headache. The neck was not stiff. There was no facial weakness. The throat was normal. The heart was not enlarged; there was normal sinus rhythm at a rate of 120 and there was a soft systolic murmur of grade 2 (I-VI) intensity heard along the left sternal border. The abdomen was soft without palpable organs or masses. The neurological examination revealed slight weakness of the left upper extremity associated with a marked incoordination as manifested by the finger-nose and handwriting tests. The biceps and triceps reflexes were slightly more active on the left. The remainder of the neurological examination was normal.

The urine was concentrated with a specific gravity of 1.050 but was otherwise normal. The hemoglobin was 13.0 grams; there were 11,600 WBC per cc. of which 89 were segmented and 3 were bands. The spinal fluid was clear under a pressure of 23 cm. of water, and 3 polys and 1 lymphocyte per ml., and 17 mg. % of protein. X-rays of the skull, mastoids, sinuses and chest were all normal. The spinal fluid proved sterile. Unfortunately, a blood culture was not immediately obtained and she received 3.4 grams of sulphadiazine in 1000 ml. of Beclysyl® shortly after admission. Twelve hours after admission the temperature was normal and remained that way; and therefore blood cultures, which had been ordered to be taken when the temperature rose to 103° or more, were not drawn.

She received oral sulphadiazine and Chloromycetin® and went home feeling fine after 5 days, to continue therapy for another 10 days. The day she left the hospital the hemoglobin was repeated and found to be 11 grams compared to the initial of 13.0 grams (attributed to hemoconcentration).

All went well for two weeks when she began to have a recurrence of fever. She was readmitted to the Thayer Hospital on the 13th of September, 1965.

The previously noted neurological findings which had cleared up during her previous hospitalization had not recurred. The only significant change in the physical examination was the heart murmur which was now of grade 4 (I-VI) intensity. During the first day the temperature ranged between 104° and 106°. The change in the heart murmur alerted the physician to the possible diagnosis and 5 blood cultures were drawn, all of which grew out a coagulase-positive staphylococcus. The initial hemoglobin was 11.0 gm. and fell to a low of 8.2 gm. three days later. During this time splinter hemorrhages were

found under the fingernails and petechiae appeared on the conjunctivae, the hands and feet. There was tenderness in the left upper quadrant but a definite spleen edge could not be felt.

Therapy, which was begun two days after admission when the cultures grew out, consisted of 40 million units of aqueous Penicillin G i.v. with "piggy-back" injections of Staphcillin® 1.0 gram every 6 hours. Streptomycin 1.0 gm. i.m. twice daily was given for 3 days. Benemid® 0.5 gm. four times daily had to be stopped after 4 days because of gastric distress.

The course was one of almost immediate improvement in well-being and appetite. The temperature became normal on the fourth day and stayed that way. Petechiae faded out over the first week. On the fourth day the patient's serum inhibited growth of the organism in a dilution of 1:4096. Blood cultures drawn after this day proved sterile. The electrocardiogram and chest x-ray were normal.

A sample of the organism was sent to the Bristol Labs. in Syracuse, N. Y. where testing showed it to be sensitive to the new oral penicillin compounds. In spite of this, it was felt that, due to the possible serious consequences of failure to sterilize the heart, i.v. therapy should be continued and it was for 26 days. At this point she was given Prostaphlin® orally 1000 mg. every 6 hours for 7 days, then 500 mg. every 6 hours for 7 days. The etiology of the underlying heart disease has not yet been definitely established. However, from the location of the murmur and its quality, an intraventricular septal defect appeared the most likely diagnosis. It is planned to establish the diagnosis at a later date at the Surgical Diagnostic Clinic of the Maine Medical Center.

At the time of her discharge the hemoglobin was 11.6 gm., having climbed from a low of 8.2 gm. with no therapy other than the antibiotics mentioned. Three weeks after discharge C.F. felt fine and was planning to return to school.

Discussion

This case of bacterial endocarditis presented itself with a localized ataxia of the left hand, doubtless due to a small septic embolus. The diagnosis was not suspected at first since there was insufficient evidence to diagnose clinical heart disease. However, when the heart murmur became more intense, the hemoglobin dropped and petechiae made their appearance, the clinical evidence made the diagnosis obvious.

Patients with bacterial endocarditis can be divided into two main groups. The younger are usually affected by rheumatic or congenital heart disease. Older patients are usually debilitated, have had recent surgery or a major septic condition and develop an endocarditis on an otherwise previously normal heart valve. The streptococci and staphylococci make up 90% of the offending organisms in bacterial endocarditis. The other cases can be caused by a wide variety of bacterial pathogens and cases have even been described due to candida and other mycoses.

In a recent series, Blount found the incidence of bacterial endocarditis was one per 979 hospital admissions (excluding psychiatric and newborn admissions). The average duration of symptoms was 2.2 months. Out of 89 cases 56% were men and 44% were women ranging in age from 5 to 70 years. Fifty-eight percent had rheumatic heart disease and twenty-three congenital heart disease. The initial survival rate was 80%. However, at the end of 5 years in most series, there is a mortality rate of 35 to 50%. Many of these late deaths are due to intractable heart failure resulting from irreparable damage to the aortic valve. Friedberg emphasizes duration of symptoms in relation to survival, claiming that a duration under 2 weeks has 100% survival, while a duration of over 2 months have only a 56% survival. On the other hand, Vogler noted the reverse and found better survival in those whose symptoms had been present longer while Blount found duration of symptoms to have no influence on survival.

Treatment should be given for at least 4 weeks when strains of organisms sensitive to Penicillin are involved, and up to 6 weeks when they are resistant. Penicillin G in doses of 10 to 100 million units per day is recommended along with Streptomycin or one of the new penicillinase-resistant penicillins. There are various techniques of serial dilution being used (see Blount) to determine the efficacy of inhibition of the infecting bacterium. Oral treatment, while not generally recommended, has been used successfully (see Campeau and Lefebvre).

Summary

A case of bacterial endocarditis has been presented. It was treated in a small community hospital. A short follow-up period suggests treatment was successful. A brief review of the literature has been included.

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14 Gilman Street, Waterville, Maine

You Need Continuing Education — Continuing Education Needs You

RICHARD T. CHAMBERLIN, M.D.

Medical education at all levels is under increasingly close and severe scrutiny and reevaluation. The problems involved are ever mounting and they will not be repeated here as they have recently been well documented by Dr. J. M. Hayman in his address to the 112th Annual Session of the Maine Medical Association in June of 1965 and published in this journal in October, 1965.[1] This article is intended only to underscore some of these problems as they apply to those physicians who are currently involved in the active practice of medicine and are in search for a solution to their own educational needs. Much of this report is based on ideas and comments expressed at the Postgraduate Conference on Medical Education in the Hospital sponsored by the Department of Postgraduate Medical Education of the University of Colorado School of Medicine held at Estes Park, Colorado on October 14-16, 1965.

One cannot help but be disturbed by current events in medical education when one reads the words of Dr. Hayman on the one hand, and yet becomes aware of certain established trends affecting medical education on the other.

One of these trends was graphically illustrated by Dr. James Campbell, Professor of Medicine at the University of Illinois and member of the AMA Internship Review Committee, in his report at the Colorado Conference entitled *The Future of the Internship.* In spite of the fact that most medical educators agree on the need to develop programs designed to train family physicians, Dr. Campbell points out that at the present time the future and structure of the internships seems to be in the process of being molded by the demands of the prospective interns themselves. In the fierce competition for good men which directors of internships and residencies find themselves, the tendency is to fashion the programs to fit these "consumers" demands in order to attract the best men. It is Dr. Campbell's thesis that it is these demands which have been a major factor in the rise in the number of so-called straight programs and the fall in the number of rotating or general training programs. The disturbing points here, however, are found when one tries to analyze what factors the "consumer" demands themselves are based on. Of course many of the prospective applicants are motivated by the desire to study and train under good teachers and recognized authorities. However, one is also aware that higher pay and all manner of fringe benefits, such as free babysitting and no nights or weekends on call, also play a significant role in the applicant's decision to apply for one program as opposed to another. This has recently been carried to a particularly dangerous extreme in one large and well recognized teaching hospital on the West coast. In this hospital the house staff is reported to have donated a sum of money to hire a lawyer who then took the young doctor's demands for such pay increases and fringe benefits to a court of law. The result of this action was that the hospital was obliged by court order to honor some of these demands. The impact of this decision has made other nearby hospitals meet similar demands or otherwise suffer the possibility of losing some of the more desirous candidates' application. It is difficult enough to plan an educationally rewarding teaching program not to have its success threatened by such mundane trivia. If one were fortunate enough to plan a successful program for the teaching of total patient care as envisioned by Dr. Hayman, how is one to combat this apparently rising trend of young physicians in training to seek positions not by their standards of excellence but by what is offered in terms of personal comforts and conveniences? What motivation will such physicians have when they are in practice towards their own continuing education?

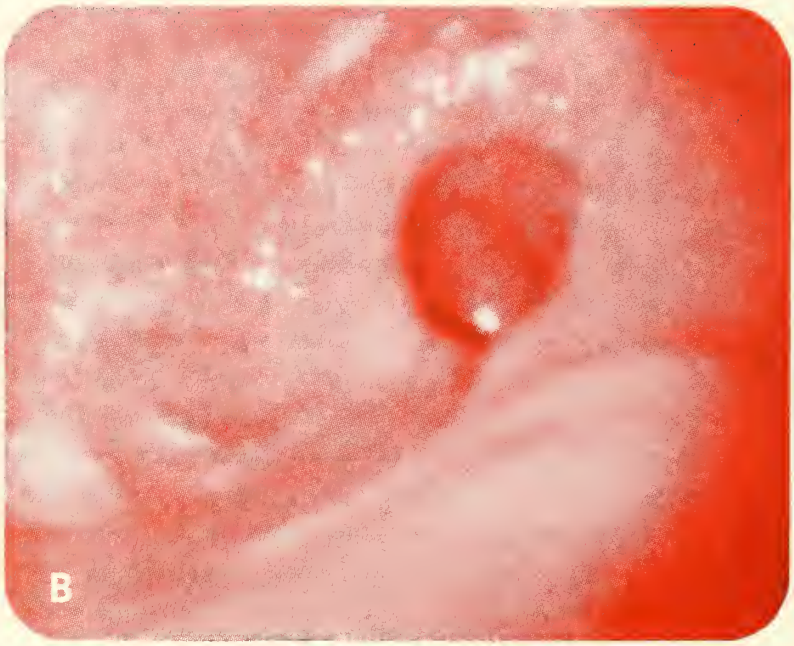
Another wind bearing ill will affects us on the scene of continuing education for the practicing physician. In a recent malpractice case a midwestern State Court upheld a verdict for the plaintiff which also enjoined the hospital Board of Trustees as well as the practitioner involved. The contention was that the Board of Trustees failed in its responsibility to insure that the physician involved kept abreast of recent advances in the field of medicine involved in the case. If the principles involved in this case are followed to their logical conclusion physicians can expect still another outside force becoming increasingly powerful in affecting their professional lives. Perhaps it is wrong to say that this is a disturbing trend or to accuse the courts of bearing "ill will," yet it does seem a shame that motivation for continued education has to be stimulated in this fashion.

As the Colorado Conference dealt with the problem of continuing education for the practicing physician, it considered four related questions. First, who needs continued education? Second, what is a non-teaching hospital? Third, who is responsible for continued education? Fourth, what are the techniques which are best suited for continued education? Everyone seems to be in agreement on the answers to the first three of the questions. All physicians are in need of continued education. There is no such thing as a non-teaching hospital - all hospitals are capable of teaching something. The physician himself is responsible for his own

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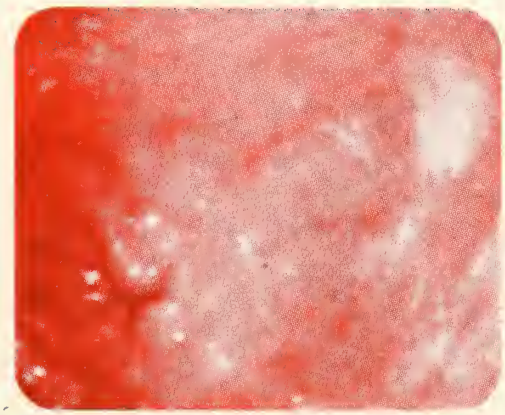


Which Is Pyloroplasty with Vagotomy?
Which Is Pro-Banthine?



Photographs—Harry Barowsky, M.D., Lawrence Greene, M.D., and Robert Bennett, M.D., from a Scientific Exhibit presented at the Annual Meeting of the American College of Gastroenterology, Bar Harbour, Florida, Oct. 24-27, 1965.

Another example of
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a true anticholinergic in action



Normal relaxed pyloric antrum; contracted pylorus (pyloric fleurette)

The true anticholinergic values of Pro-Banthine have never been so graphically realized as they are with the recent development of fibergastroscopy and the intragastric camera.

Pro-Banthine consistently produces complete relaxation and immobility of the stomach with a dose of only 6 to 8 mg. intravenously. This is less than half the usual dose orally.

Atropine, on the other hand, required 0.8 mg. intravenously, or twice the normal dose, to achieve a similar effect. This high dose of atropine resulted in expectedly adverse side effects.

Pro-Banthine, in minimal dosage, produces effects similar to pyloroplasty and vagotomy without the disadvantages of permanent post-vagotomy sequelae.

The intragastric photograph A is of a patient who has had pyloroplasty with vagotomy. Photograph B is of a patient given 6 mg. of Pro-Banthine.

Indications: Peptic ulcer, functional hypermotility, irritable colon, pylorospasm and biliary dyskinesia.

Oral Dosage: The maximal tolerated dosage is usually the most effective. For most *adult* patients this will be four to six 15-mg. tablets daily in divided doses. In severe conditions as many as two tablets four to six times daily may be required. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg., as prolonged-acting tablets of 30 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

Side Effects and Contraindications: Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. Pro-Banthine is contraindicated in patients with glaucoma, severe cardiac disease and prostatic hypertrophy.

SEARLE

Research in the Service of Medicine

continuing education. The problem then resides in the answer to the fourth question - what techniques *are* most effective?

In considering the answer to this last question, one must always bear in mind what may be the most important factor in evaluating and planning any proposed technique to be used. Dr. George Robertson, Medical Director of the Bingham Associates Fund, has often pointed out that the most important commodity possessed by a practicing physician is his time. Whatever technique is proposed for use in continuing education will certainly fail if it is not economic with the physician's time. Just as educators such as Dr. Hayman and Dr. Campbell are struggling with the curriculum problems of medical education on the medical school and internship level, so should we individual physicians in practice be making an effort to discover the most effective and least time consuming methods of insuring our own self advancement.

This is not to imply that the problems of medical education on the undergraduate and immediate postgraduate level are completely divorced from those on the level of the later postgraduate years. The so-called town and gown syndrome should not be allowed to infect the body of medical education. That the medical school-research center has much to offer the practicing physician is obvious. But, so too does the practicing physician have a great wealth of knowledge which should be of interest to the medical center in the form of large numbers of practical day to day clinical observations made on patients in his practice. Much of our basic medical knowledge has come to us through the years from the study of patients. Most educators still agree that the most lasting knowledge an individual physician has at his fingertips has come from his own experience with a patient at the bedside. Here is a wealth of learning material we deal with every minute of our professional lives. Now with the marvels of electronic computers we can record and analyze data made by simple clinical observation. Use of such data collected by the physician and studied by himself, as well as the medical center, could easily be used in the physician's own self improvement as well as to discover weak points in the teaching of clinical subjects in medical school especially in the concept of teaching total patient care.

One current methodology using these principles is available in the Professional Activity Study (PAS). Doctors Virgil Slee and Kenneth Williams demonstrated at the Colorado Conference the ease with which clinical observations may be collected, stored, analyzed, and returned for use in teaching. Examples they have used to demonstrate the value of this system include the discovery by one hospital which was studying the high incidence of one unit transfusions in that institution that the laboratory calibration of the hemoglobinometer was in error. This has led to a large number of falsely low hemoglobin readings which in turn had caused many physicians in that institution to order unnecessary transfusions.

It would seem that by the use of such a system many time consuming committee functions could be coincidentally discharged while the physicians involved were in the process of self-evaluation and self-instruction. The tissue audit committee could not only review their charts, but would be simultaneously discovering weak points in the way their particular staff was handling certain clinical problems. This information could then be passed on to the director of medical education to assist him in planning staff educational programs centered on that particular problem using the staff's own patient material. This type of continuing education should have greater value in terms of a lasting learning experience than the merely arbitrary scheduling of didactic lectures on subjects which may have no particular interest in the mind of the staff at the moment.

This or similar methods would seem to have limitless applications to medical education. Data on the natural history of disease and on ambulatory patient care could be collected, computerized, and fed back from the field to the medical center to assist in the planning of future undergraduate curricula. The analysis of the data by the increasing numbers of super specialists in the research centers could lead to a new knowledge to be fed back to the practitioner. Standards of patient care in one locale can be compared to the combined experiences of large numbers of similar hospital experiences and serve as guidelines to improve certain of these standards on any local level. The ultimate goal of this methodology could be en extension of it to the development of similar methods by which an individual physician might be able to make and record clinical observations on his ambulatory office patients too. Such data on this large segment of patients who are never hospitalized for study is sadly lacking at present–yet without this information any planning of educational programs designed to train a family physician in total patient care is going to be sadly lacking in essential building blocks needed for success.

In summary, the field of continuing education for the practicing physician, while it has its perplexing problems, also has some exciting possibilities within easy reach. The dissemination of our rapidly expanding medical knowledge from the medical center to the practicing physician still presents an unsolved problem. However, the concept of making the practicing physician an important partner in the research on total patient care as outlined above could not only serve to motivate him for his own continuing education but also make him a most important contributor to the ultimate goal of all medicine - the best possible medical care for all mankind.

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14 Gilman Street, Waterville, Maine

Maine Heart Association Notes

116 State Street, Augusta, Maine

Hypertension and Unilateral Renal Disease
The Usefulness of Modified Intravenous Urography

"In recent years, the intravenous urogram has become increasingly important in the detection of patients with potentially curable renovascular hypertension . . . Renal ischemia has both morphologic consequences, mainly renal atrophy, and functional consequences, increased reabsorption of urinary sodium and water by the renal tubules resulting in a reduced output of more concentrated urine by the ischemic kidney. To better identify these changes, the intravenous urogram . . . has been modified by the use of rapid-injection technique, the elimination of abdominal compression, and the addition of minute sequence films. Observed differences in the appearance time of . . . contrast material . . . and difference in its density reflect inequalities in urine output and concentration by the two kidneys . . . utilizing this rapid sequence type of intravenous urography, in a recent series of 200 hypertensive patients we have been able to suspect renal ischemia with greater than 90 per cent accuracy in patients with significant major renal artery obstruction as shown by subsequent aortography and other function tests in the same group of cases.

This modification of the urogram is safe. It requires the same time as an ordinary intravenous urogram. It can be performed in any radiology department without special equipment. With experienced interpretation it yields relatively few false-positive results . . ."

Submitted by Jacob B. Dana, M.D.
Reference: Fleming, R. J., et al, Circulation, Vol. 32, Page 682-685, 1965.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Plant Poisoning Umbelliferae (parsley family)

E. W. CAMPBELL, DR. P.H.*

CICUTA MACULATA L. WATER HEMLOCK

A stone mason working with a road crew picked up a root which he tentatively identified and thought to be a root of the plant known as ginseng, proceeded to chew a part of this root, believing it to have medicinal properties, and a plant which is normally used in China as a medicinal, or similar preparation. According to the report, in a very few minutes he showed symptoms of inebriation, then was confused and within a very short time developed abdominal pains, vomited profusely, and finally had a series of generalized convulsions which cumulated in coma and cardiac arrest.

Specimens of the root in question and accompanying foliage was promptly collected and sent by messenger to the toxicology section of the laboratories of the Division of Sanitary Engineering, State Department of Health and Welfare, Augusta, Maine. An article appeared in an evening paper that this person's death had been caused by eating a plant which was thought to be Jimson weed, on the basis of the information furnished by persons working with the victim. This was only suspect, and not enough information was given to correctly identify the plant, and it was only until a sample was received at the laboratory that reference to standard textbooks on poisonous plants indicated that it was not Jimson weed (Datura stramonium L.) which is also known as Jamestown weed, and occasionally as thorn apple.

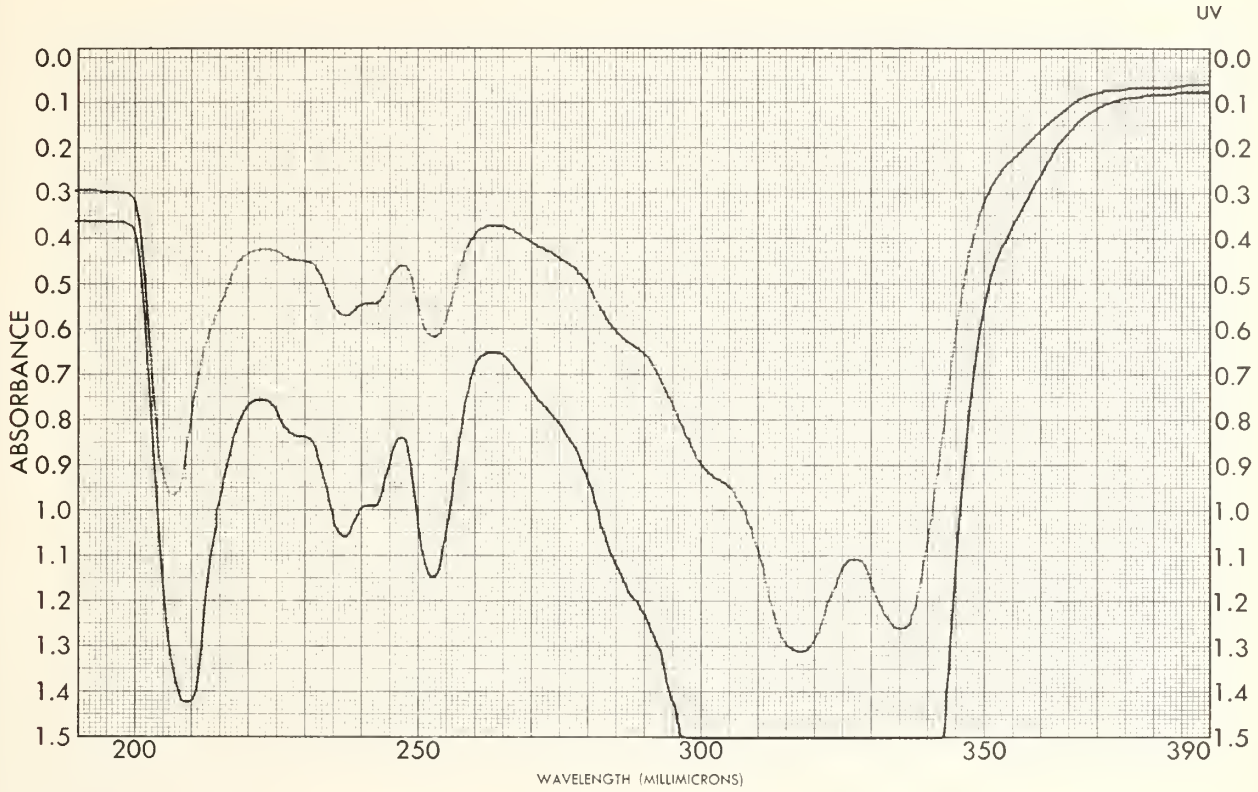
Apparently there has been very little work done on actual methods of analysis for poisons of this nature, and a search of the literature disclosed very little information. "Poisonous Plants of the United States," indicate that the poisonous principle of this plant, Cicuta maculata L., is a substance known as cicutoxin, a clear brown sticky substance with an acid reaction which is soluble in alcohol, chloroform, ether, and dilute alkalies. The roots and root stalks are the most poisonous parts of the plant, but apparently all parts may contain some of the poison, especially in the early stages of



growth. Cicuta is described as containing the most virulent species in the United States, and poisoning has been reported a number of times in human beings. Children and even adults have eaten the fleshy roots by mistaking them for parsnips, artichokes, or other roots, with serious or fatal results. Among the domestic animals most losses are encountered among cattle, however, horses, sheep and swine are sometimes killed. It is very probable that the plants are poisonous at all seasons. A greater number of cases of poison in the spring may be accounted for by several conditions.

Cicuta grows primarily in wet or swampy ground, frequently near streams, or in swales in pastures. Such plants usually become green before the rest of the pasture, and when animals are turned out to pasture they go to these places and while eating the tops of the

*Director, Division of Sanitary Engineering (Retired on December 17, 1965)



WATER HEMLOCK EXTRACT

SAMPLE Alcohol Extract — vacuum evaporated, ether washed with NaOH, evaporated, paper chromatography (Butyl alcohol, acetic acid, water) ORIGIN SOLVENT methyl alcohol	CURVE NO. CONC. diluted to give curve (conc. from paper chrom. not known) CELL PATH 1 cm. REFERENCE Journal Chem. Society (Lond.) 309 (1953)	SCAN SPEED slow SLIT 25 REMARKS	OPERATOR R.C.E. DATE Oct. 6, 1965

cicuta they also pull out the roots which are apparently more poisonous. The quantity of cicuta necessary to cause death probably varies with the season and the age of the plant. A piece of root about the size of a walnut is said to be enough to cause the death of a cow. At any rate, only a very small quantity is necessary to cause fatal poisoning. The symptoms of cicuta poisoning as indicated are similar to those which have been reported in the case in question, and apparently this case followed the usual course which has previously been observed.

Treatment as indicated in the book referred to is to give an efficient emetic, and if possible followed by a cathartic. Vomiting must be induced immediately, and if prompt enough and effective it is possible that the patient may recover. If there is any lapse of time, however, and symptoms have already begun, the prognosis is extremely poor as fatal results develop very quickly. In the textbook previously referred to it has been suggested that in cases of poisoning of domestic animals that treatment is considered practically hopeless, but in some cases if very little of the plant has been consumed recovery may occur.

Further search of the literature found some work on the identification of Cicutoxin which was published in the *Journal of the Chemical Society* in London in 1953. This work done by Anet, Lythgoe, Silk, and Tripett of Cambridge, England, indicates that toxic principles of Cicutoxin have the following formula:

$HOCH_2CH_2CH_2\ (C{\equiv}C)_2(CH{=}CH)_3CHOHCH_2CH_2CH_3$

All-*trans*-heptadeca-8: 10 : 12-triene-4 : 6-diyne-1 : 14-diol

Using this work as a reference we proceeded to determine whether or not we could isolate and identify this substance.

The toxic substance Cicutoxin in water hemlock is readily decomposed by exposure to heat, light and air. All three must therefore be avoided in extracting it from the root. An ether extract of the root was washed in (1 N) one normal sodium hydroxide, filtered and evaporated to dryness under vacuum.

An infrared curve was run immediately. The infrared absorption varies greatly with time exposed to air, heat and light. However, we were able to get bands at 2212, 2128, 1634, 1603, and 996 in ($CHCl_3$) chloroform all in agreement with work done at Cambridge.

An ultraviolet curve was also prepared from the ether extract that had been purified by paper chromatography, in which the solvent was butyl alcohol, acetic acid and water (100:10:32). Areas on the paper that

showed the greatest absorption under ultraviolet light were re-extracted for use in the ultraviolet spectra determination. λ max. were at 335.5, 318, 252, 242 and inflections at 303 and 286, all in agreement with the work done at Cambridge, England.

A copy of the ultraviolet curve also has been included so that further investigators may wish to verify or duplicate this work in the identification of this or similar specimens.

In view of the resemblance of the curves to those described by the article in the *Journal of the Chemical Society* in London, it is believed that the toxic principle of the plant in question, without doubt, was that of Cicutoxin.

REFERENCES

λ max. = Ultraviolet wave length maximum.

1. Poisonous Plants of the United States by Walter Conrad Muenscher.
2. Anet, E. F. L. J., B. Lythgoe, M. H. Silk, & S. Trippett. Oenanthotoxin and Cicutoxin. Isolation and Structure. *J. Chem. Soc.* (London) 1953: 309. 1953.
3. Accidental Poisoning in Children by The Committee on Accident Prevention, American Academy of Pediatrics by Edward Press, M.D., M.P.H.
4. Poisonous Plants of United States and Canada by John M. Kingsbury.

Elmer W. Campbell, B.S., M.P.H., Dr.P.H., F.A.P.H.A., F.R.S.H., Director, Division of Sanitary Engineering, Maine State Department of Health and Welfare

and

Robert C. Ericson, B.S., Chemist III, Toxicology Laboratory, Maine State Department of Health and Welfare.

JOIN MEDICARE?

Of those eligible, many have still not signed up for Medicare. As of the first of February, there were still just a little over ten million who had agreed to participate. There appears to be a serious lack of understanding among the "eligible" elderly citizens. Here are some facts:

- As of February 1, 1966, only fifty-six per cent of those eligible have signed up.
- The eligible citizens must sign up before March 31, 1966. This includes all those who were 65 before January 1, 1966.
- For those reaching 65 on January 1, 1966 or later, the seven month enrollment period runs from three months before the month of your birthday until three months after it.
- Those eligible citizens who do not enroll before March 31, 1966 will not get another chance until October 1, 1967 and they will not be eligible for benefits before July of 1968.
- Other health insurance policies will no doubt be tailored to cover those services not covered by the Social Security Plan. Practically all private health insurance carriers will offer new policies built around the Medicare Plan.
- Urge your patients to enroll in the Medicare Plan. If they have lost the card which was sent to them by the Social Security Administration, a call to the nearest Social Security Office will set the wheels in motion for another one.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

American Cancer Society—1966 Scientific Session

May 11, 1966 — St. Francis Hotel — San Francisco, California

Morning Session — 9:00 a.m.-12:30 p.m.

UTERINE CANCER

The Present Status of Cytology in Uterine Cancer
Leopold G. Koss, M.D., Memorial Hospital for Cancer and Allied Diseases, New York, New York

The Optimum Management of a Patient with a Positive Cervical Cytology Report
Tommy N. Evans, M.D., Wayne State University School of Medicine, Detroit, Michigan

RECTAL AND COLON CANCER

Current Procedures in the Detection and Diagnosis of Colon and Rectal Cancer
H. Marvin Pollard, M.D., The University of Michigan Medical Center, Ann Arbor, Michigan

Modern Treatment of Colon and Rectal Cancer
J. Englebert Dunphy, M.D., University of California School of Medicine, San Francisco, California

LUNG CANCER

The Control of Cigarette Smoking in the U.S.A.
Senator Maurine B. Neuberger, The United States Senate, Washington, D.C.

The Early Diagnosis of Lung Cancer
Thomas Carlile, M.D., The Mason Clinic, Seattle, Washington

BREAST CANCER

Advances in the Early Diagnosis of Breast Cancer
Thomas W. Botsford, M.D., Harvard Medical School, Boston, Massachusetts

The Breast Cancer Treatment Controversy
Robert C. Hickey, M.D., University of Wisconsin Medical Center, Madison, Wisconsin

Afternoon Session — 1:30 p.m.-4:00 p.m.

ORAL CANCER

Oral Lesions of Neoplastic Significance and Oral Cancer
Sol Silverman, D.D.S., University of California School of Dentistry, San Francisco, California

Management of Oro-Pharyngeal Cancer
Arthur G. James, M.D., Ohio State University Medical Center, Columbus, Ohio

SKIN CANCER

Early Recognition and Diagnosis of Skin Cancer
Herbert L. Traenkle, M.D., University of Buffalo School of Medicine, Buffalo, New York

The Treatment of Skin Cancer
Richard H. Jesse, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas

SPECIAL ADDRESS

Premises and Prospects in Cancer Research
Paul Weiss, Ph.D., The University of Texas Graduate School of Biomedical Sciences, Houston, Texas

Sessions are open to all members of the Medical and Dental Professions and Students. No advance registration or registration fee.

Necrologies

CHARLES L. CRAGIN, M.D.
1876-1965

Charles L. Cragin, M.D., 89, of Portland, Maine died on November 18, 1965. He was born in Norway, Maine on March 2, 1876, son of Jeremiah D. and Susie Sowes Cragin.

Dr. Cragin graduated from Norway High School, received his medical degree from Bowdoin Medical School in 1904 and interned at the Maine General Hospital and the Maine Eye and Ear Infirmary. He practiced medicine in Portland for sixty years, was the Portland City physician in 1906 and for many years served the Maine Central Railroad as physician and surgeon.

Dr. Cragin was an Honorary member of the Cumberland County Medical Society and the Maine Medical Association, having received a 50-year pin in 1954, a 55-year pin in 1959 and a 60-year pin in 1964. He was also a member of the American Medical Association, the American College of Surgeons, the American Association of Railroad Surgeons, the Oxford Lodge, AF and AM, the Maine Consistory, 32nd degree, Scottish Rite, Valley of Portland, Mt. Vernon Chapter, RAM, Portland Council, R and SM and the Portland Lodge of Elks.

Surviving are two sons, Charles L. Cragin, Jr., Portland and Major Robert P. Cragin, USAF, stationed in Southeast Asia; a daughter, Mrs. Ralph C. L. Lewin, Portland and several grandchildren, great grandchildren, nieces and nephews.

HENRY PEARSON, M.D.
1879-1965

Henry Pearson, M.D., 86, of Brownfield, Maine died on November 30, 1965. He was born in Tuscaloosa, Alabama on March 14, 1879, son of Dr. William G. Beatty and Julia Penn Snow Pearson.

Dr. Pearson graduated from the University of Alabama in 1899 and received his medical degree from Cornell University Medical School in 1903. He did postgraduate work at Bellevue Hospital in New York from 1903 to 1905. He practiced medicine in New York City from 1905 to 1928 and then moved to Brownfield where he practiced until his retirement.

Dr. Pearson was an Honorary member of the Oxford County Medical Society and the Maine Medical Association, having received a 50-year pin in 1953, a 55-year pin in 1958 and a 60-year pin in 1963. He was also a member of the American Medical Association, the Medical Association of Alabama, the Medical Society of Massachusetts and was a fellow of the New York Academy of Medicine.

Surviving are a nephew, Major General Alden K. Sibley, stationed in Germany; two nieces, Mrs. Sumner W. Atkins, Newington, Connecticut and Miss Julia Pearson Sibley, New York City.

SABRO TASHIRO, M.D.
1910-1965

Sabro Tashiro, M.D., 55, of Gardiner, Maine died on December 1, 1965. He was born in New Haven, Connecticut on March 1, 1910, son of Ayiro and Nao Tashiro.

Dr. Tashiro graduated from the University of Cincinnati in 1935 and received his medical degree from the University of Cincinnati College of Medicine in 1937. He interned at the Cincinnati General Hospital from 1937 to 1938 and served residencies in pathology and surgery at the Cincinnati General Hospital and Bethesda Hospital from 1938 to 1944.

Dr. Tashiro was a surgeon at the Togus Veterans Administration Center from 1948 to 1952 and then moved to New York where he practiced surgery at the Batavia Veterans Administration Hospital from 1953 to 1958. He moved to Gardiner in 1959 following his retirement.

He was a member of the Kennebec County Medical Association, the Maine Medical Association and the American College of Surgeons.

Surviving are his widow, Mrs. Beverly Haresign Tashiro; four brothers, a sister and several nieces and nephews.

County Society Notes

KNOX

Twenty-three members and one guest were present at the meeting of the Knox County Medical Society which was held on December 7, 1965 at the House of 1800 in Glen Cove, Maine.

Mr. Richard F. Nellson, Executive Director of the Associated Hospital Service of Maine, spoke on the Medicare law prior to the official meeting. He explained how the presently existing government agencies will be incorporated into the Medicare set up and gave reasons why he feels that Blue Cross-Blue Shield would be the best qualified fiscal intermediary.

Johan Brouwer, M.D. strongly urged to have the intensive care form changed and improved.

The members were notified of the existence of two booklets, one concerning itself with Area Wide Health Facility Planning, the other one a Review of Hospital Utilization. The secretary was requested to order more copies of the latter. The members were informed that the Food and Drug Administration is collecting data on untoward drug reactions.

Edward K. Morse, M.D. as a member of the Health Insurance Committee, reported relative to a Blue Shield investment program for physicians.

The members of the society voted to approve Blue Cross-Blue Shield as fiscal carrier for Medicare.

The nominating committee presented the following slate of officers which was approved:

President, Frank W. Kibbe, M.D., Lincolnville
Vice-President, Peter H. Holz, M.D., Camden
Secretary-Treasurer, William W. Ward, M.D., Rockland

PETER H. HOLZ, M.D., *Secretary*

HANCOCK

The annual meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on December 8, 1965.

The following officers were elected for 1966:

President, Bradley E. Brownlow, M.D., Blue Hill
Vice-President, Robert D. Wilson, M.D., Bar Harbor
Secretary-Treasurer, Russell G. Williamson, M.D., Blue Hill

Delegates to the Maine Medical Association House of Delegates: Drs. Herbert T. Wilbur, Jr., Southwest Harbor and Russell M. Lane, Blue Hill. Alternates: Drs. Arthur M. Joost, Jr., Bucksport and Elizabeth E. Williamson, Blue Hill

Censors, Drs. Charles H. Knickerbocker, Bar Harbor (3 yrs.), John D. McIntyre, Ellsworth (2 yrs.) and W. Edward Thegen, Bucksport (1 yr.)

RUSSELL G. WILLIAMSON, M.D., *Secretary*

KENNEBEC

The Kennebec County Medical Association held its annual meeting on December 9, 1965 at the Augusta State Hospital. Mr. Richard F. Nellson of the Associated Hospital Service of Maine spoke to the Association about the role of the fiscal intermediary in the supplementary medical insurance plan. Mr. Nellson pointed out the reasonable and desirable features of having the Associated Hospital Service of Maine act as the fiscal intermediary for the "Medicare" program here in the State of Maine.

The nominating committee, consisting of the three most recent past presidents, Drs. Loring W. Pratt, Kenneth W. Sewall and Brinton T. Darlington, presented the slate of officers for the year 1966 which was elected unanimously.

President, Allan J. Stinchfield, M.D., Augusta

Vice-President, Robert L. Ohler, M.D., Togus

Secretary-Treasurer, Lucien F. Veilleux, M.D., Waterville

Delegates to the Maine Medical Association House of Delegates: Drs. John M. H. Barnard and Francis J. O'Connor, both of Augusta, Earle M. Davis, Paul A. Jones, Jr. and Valentine J. Moore, all of Waterville. Alternates: Drs. Richard T. Chamberlin and Richard L. Chasse, both of Waterville, Theodore M. Russell and John H. Shaw, both of Augusta, and Francis A. Spellman, Togus

Councilors, Drs. Richard H. Dennis and Vaughn R. Sturtevant, both of Waterville and Joseph R. Crawford, Augusta

Grievance Committee, Drs. Oakley A. Melendy, Augusta, George 1. Gould, Richmond and Edmund N. Ervin, Waterville

Manpower Procurement and Civil Defense Committee, Drs. Albert A. Poulin, Waterville, M. Tieche Shelton, Augusta and Clarence R. McLaughlin, Gardiner

John C. Patterson, M.D., Superintendent of the Augusta State Hospital, introduced a panel of speakers who discussed various psychiatric activities as carried on throughout the State of Maine. Richard A. Levy, M.D. of Portland spoke of the Mental Health Clinic which is being held at the Maine Medical Center and which is sponsored by the State of Maine, the United Fund and the Maine Medical Center itself.

Allen I. Saunders, M.D. discussed the changes which have been brought about by the adoption of the Durham Rule in handling medical-legal and psychiatric problems. Walter Rohm, M.D. discussed the activities of the outpatient clinic, now in its second year, at the Augusta State Hospital.

Dr. Denison then relinquished the presidency to Dr. Stinchfield who adjourned the meeting.

EARLE M. DAVIS, M.D., *Secretary*

CUMBERLAND

Ninety-five members and guests attended the annual meeting of the Cumberland County Medical Society which was held at Valle's Steak House in Portland, Maine on December 16, 1965.

Formal presentations, followed by a question and answer period, were made by representatives of two of the potential carriers of Part B of Medicare; Mr. Robert Roberts of the Union Mutual Life Insurance Company and Mr. Richard F. Nellson of the Associated Hospital Service of Maine. After the discussants left the meeting, the membership voted to allow Albert Aranson, M.D. to use his judgment as to how to vote at the next meeting of the Health Insurance Committee of the Maine Medical Association.

William L. MacVane, Jr., M.D. reported that he had investigated the American Association of Medical Assistants and had found the organization to be approved by the American Medical Association as being a legitimate and worthwhile organization. The membership voted in favor of a Maine Chapter.

Philip G. Good, M.D. delivered his annual report of the Public Relations Committee and Edward G. Asherman, M.D. reported on progress by the Medical-Legal Liaison Committee, with definite headway toward solution of common problems.

An audit committee was appointed with John F. Gibbons, M.D. as chairman and Drs. David S. Wyman and George F. Sager as members. The secretary gave a brief annual report and Alvin A. Morrison, M.D. summarized the years' activities in his report as retiring president.

The nominating committee consisting of Drs. Charles R. Geer, Donald F. Marshall and G. Hermann Derry presented a slate of officers for 1966 which was approved:

President, Alvin A. Morrison, M.D., Portland

Vice-President, Frederick B. Clark, M.D., Portland

Secretary-Treasurer, Stanley B. Sylvester, M.D., Portland

Delegates to the Maine Medical Association House of Delegates: Drs. Charles R. Glassmire, Portland, Lloyd G. Davies, Fryeburg, Alvin A. Morrison and Frederick B. Clark (2 yrs.); Drs. Charles R. Geer, Howard P. Sawyer, Maurice VanLonkhuyzen, Philip P. Thompson, Jr., David K. Lovely and Clement A. Hiebert, all of Portland (1 yr.). Alternates: Drs. Sidney R. Branson, South Windham, Kirk K. Barnes, Brunswick, John F. Gibbons and Robinson L. Bidwell, both of Portland (2 yrs.); Drs. Raphael F. Turgeon, Westbrook, Harold N. Burnham, Gorham, Hugh P. Robinson, Irving J. Poliner and Louis A. Ciampi, all of Portland (1 yr.)

Public Relations Committee, Drs. George I. Geer, Jr., Chairman, Philip G. Good and Robert V. Lorimer, all of Portland

A film entitled "How to Watch Pro Football" completed the evening.

STANLEY B. SYLVESTER, M.D., *Secretary*

LINCOLN-SAGADAHOC

The Lincoln-Sagadahoc County Medical Society met at The Ledges in Wiscasset, Maine on December 21, 1965 with the President, Carl R. Griffin, Jr., M.D., presiding.

John F. Dougherty, M.D., President of the Maine Medical Association, mentioned that the American Medical Association is planning to raise dues to $70.00 a year and asked that this society discuss how the Maine Medical Association delegate to the AMA should vote on this matter. Dr. Dougherty then mentioned several other reports from the Council of the Maine Medical Association which did not require action. The nebulous plans of the Federal program for Heart Disease, Cancer and Stroke were discussed.

The guest speaker, Donald P. Dressler, M.D. of Cambridge, Massachusetts, spoke on the control of burn wound sepsis.

GEORGE W. BOSTWICK, M.D., *Secretary*

YORK

The annual meeting of the York County Medical Society was held at the Henrietta D. Goodall Hospital in Sanford, Maine on January 5, 1966.

The following slate of officers were elected for 1966:

President, Walter R. Peterlein, Jr., M.D., Springvale

Vice-President, Eugene P. Wolfahrt, M.D., Saco

Continued on Page 47

Letter to the Editor

Re: "Psychiatry and Criminal Responsibility" by Peter W. Bowman, M.D. which appeared in the January, 1966 issue of your Journal

Dr. Bowman sums up succinctly some of the major problems affecting adequate use of psychiatric testimony by the courts but there are aspects of his proposed solutions that call for more discussion.

1. Granted that we should have a determination of whether or not a man did what he is accused of before the question of sanity is raised, what happens if his lawyer says "this man can't cooperate in his own defense," (an element in due process) any legal change would have to include procedures for this instance.

2. In areas where the probation office functions with reasonable adequacy, the judge has at his disposal a pre-sentence investigation of the defendant which he can use in passing sentence. Extending this investigation to include psychiatric evaluation either on an outpatient, in jail, or in hospital basis has provided judges with useable advice on disposition.

3. The judge has a series of problems of course (a) "What's the most sensible disposition?" (b) "Can I leave him in the community (on probation) and if so what kind of control and supervision can my court provide?" His move in this instance clearly depends, at least as much on the quality of his probation staff and other community services as it does on the mental condition of the defendant. (c) If the judge decides on institutionalization he should have the option of sending the man to the most suitable institution in the state be it a prison or a mental hospital or a training school etc.

4. California has taken the question of disposition out of the court and given it to a sentencing board or authority. The problem of sending the man to the most appropriate institution still remains. I know of no jurisdiction where a "mentally ill" person can be sent to an ordinary prison–even though that might be the best place for him–from a rehabilitative point of view–and vice versa with "criminals" and mental hospitals.

5. The ideal solution might be to limit the courts to a determination of whether or not an accused committed a criminal act (whether or not he can participate in his own defense) and leave to a sentencing board the problem of doing an adequate investigation and determining when necessary in what type of institution the man should be kept (and for how long). Unfortunately this kind of solution may not be consistent with some rather important essentials of a free society. The civil liberties lawyers might be able to come up with some counter proposals however.

I'm pleased that the Journal has opened its pages to these discussions of public policy.

LEON N. SHAPIRO, M.D.
Boston, Massachusetts

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COUNTY SOCIETY NOTES — *Continued from Page 45*

Secretary-Treasurer, Charles W. Kinghorn, M.D., Kittery
Delegates to the Maine Medical Association House of Delegates: Drs. Carl E. Richards, Sanford, Robert F. Ficker, Kennebunkport and Thomas Anton, Biddeford. Alternates: Drs. Irvin Dorfman, Biddeford, Stephen A. Cobb, Sanford and Kenneth E. Leigh, York
Executive Committee, Drs. Walter R. Peterlein, Jr., Eugene P. Wolfahrt, Charles W. Kinghorn, Carl E. Richards and Kenneth E. Leigh
Board of Censors, Drs. Stephen A. Cobb, Melvin Bacon, Sanford and Willard H. Bunker, York Harbor
Resolutions Committee, Drs. Stephen A. Cobb, Maurice Ross and Paul S. Hill, Jr., both of Saco
Auditing Committee, Drs. Leopold A. Viger, Biddeford, J. Robert Downing, Kennebunk and Melvin Bacon
Medical Procurement Committee, Drs. George A. Lord, Sanford, Walter R. Peterlein, Jr. and Robert F. Ficker

The guest speaker was Richard F. Nellson, Executive Director of the Associated Hospital Service of Maine, who spoke on Blue Shield and Medicare.

Paul S. Hill, Jr., M.D. spoke on matters relating to activities of the Council of the Maine Medical Association and Roger J. P. Robert, M.D. discussed matters pertaining to the Health Insurance Committee of the Maine Medical Association.

The society voted to send "Today's Health," a publication of the American Medical Association, to all high school libraries in York County.

The next meeting will be held on Wednesday, March 9 at Webber Hospital in Biddeford.

CHARLES W. KINGHORN, M.D., *Secretary*

News, Notes and Announcements

Central Maine General Hospital 1966 Postgraduate Refresher Course in Obstetrics-Gynecology

Hiebert Hall Fee: $30.00 3:00-6:00 P.M.

March 30—*Basic Female Endocrinology*
Janet W. MacArthur, M.D., Assistant Professor of Clinical Medicine, Harvard Medical School, Boston, Massachusetts

April 6—*Clinical Use of Hormones*
Robert W. Kistner, M.D., Assistant Professor of Obstetrics & Gynecology, Harvard Medical School, Boston, Massachusetts

April 13—*Erythroblastosis*
Louis K. Diamond, M.D., Professor of Pediatrics, Harvard Medical School, Boston, Massachusetts

April 20—*Current Concepts in Management of Hydatidiform Mole and Trophoblastic Tumors*
Donald P. Goldstein, M.D., Instructor in Obstetrics and Gynecology, Harvard Medical School, Boston, Massachusetts

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AN ANCHOR TO WINDWARD

April 27—*Tumors of the Ovary and Endometrium*
Charles T. Griffiths, M.D., Instructor in Obstetrics & Gynecology, Harvard Medical School, Boston, Massachusetts.

May 4—*Surgical Complications in Obstetrics*
Edwin L. Carter, M.D., Assistant Instructor in Surgery, Harvard Medical School, Boston, Massachusetts

George T. Nilson Appointed Chairman of the Maine Interagency Council on Smoking

George T. Nilson, field director of the Bingham Associates Fund, has been appointed chairman of the newly-organized Maine Interagency Council on Smoking. Members of the Council are the Maine Cancer Society, Maine Heart Association, Maine TB and Health Association, Maine Department of Education, Maine Department of Health and Welfare, National Foundation (Maine Chapter) and the Bingham Associates Fund. Two physicians, Dr. Brinton T. Darlington of Augusta and Dr. Stanley C. Beckerman of Waterville, represent, respectively, the Maine Heart Association and the Maine Department of Health and Welfare.

The Interagency Council is a subsidiary of the Health Council of Maine. The objective of the new Interagency Council is to plan and demonstrate an effective, cooperative program combatting cigarette smoking as a health hazard. A grant of $6,500 has been awarded by the Public Health Service to the Council for assistance with the project.

Young people may be a primary focus of the program but not exclusively so. It is recognized that community and family attitudes toward cigarette smoking are strong factors in youth's decision to smoke—or not. Lung cancer has reached epidemic proportions. Cardiovascular disease, emphysema, chronic bronchitis are on the march. The Interagency Council plans to spearhead the attack on cigarette smoking as a health hazard and is looking forward to the active participation and support of physicians throughout the State of Maine in this vitally important health program.

Public Health Service Project Grants for Cancer Control

What are the Objectives?—The objectives of the Cancer Control Project Grants Program are:

—to provide a mechanism for qualified individuals to develop and explore new and unique methods of cancer control,

—to demonstrate and extend the use of known protective measures to more people through their normal channels of medical care.

Who is Eligible?—Project grants may be made to the following:

—State Health Agencies
—Local Health Agencies
—Public or Private Non-profit Institutions or Agencies
—Individuals (for certain types of training)

**Examples*: educational institutions, health departments, hospitals, professional associations, welfare agencies and social agencies.

How are Applications Made?—Application forms and instructions, information on terms and conditions, and assistance in completing the forms may be obtained from the Regional Health Directors of the Regional Offices of the U.S. Department of Health, Education, and Welfare.

The Regional Office Address for Maine, Connecticut, Massachusetts, New Hampshire, Rhode Island and Vermont is: Regional Health Director, Public Health Service, Region 1, 120 Boylston Street, Boston, Massachusetts 02116.

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Indications: 'Deprol' is useful in the management of depression, both acute (reactive) and chronic. It is particularly useful in the less severe depressions and where the depression is accompanied by anxiety, insomnia, agitation, or rumination. It is also useful for management of depression and associated anxiety accompanying or related to organic illnesses.

Contraindications: Benactyzine hydrochloride is contraindicated in glaucoma. Previous allergic or idiosyncratic reactions to meprobamate contraindicate subsequent use.

Precautions: *Meprobamate*—Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage.

Benactyzine hydrochloride—Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria.

Meprobamate—Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Dosage: Usual starting dose, one tablet three or four times daily. May be increased gradually to six tablets daily and gradually reduced to maintenance levels upon establishment of relief. Doses above six tablets daily are not recommended even though higher doses have been used by some clinicians to control depression and in chronic psychotic patients.

Supplied: Light-pink, scored tablets, each containing meprobamate 400 mg. and benactyzine hydrochloride 1 mg.
Before prescribing, consult package circular.

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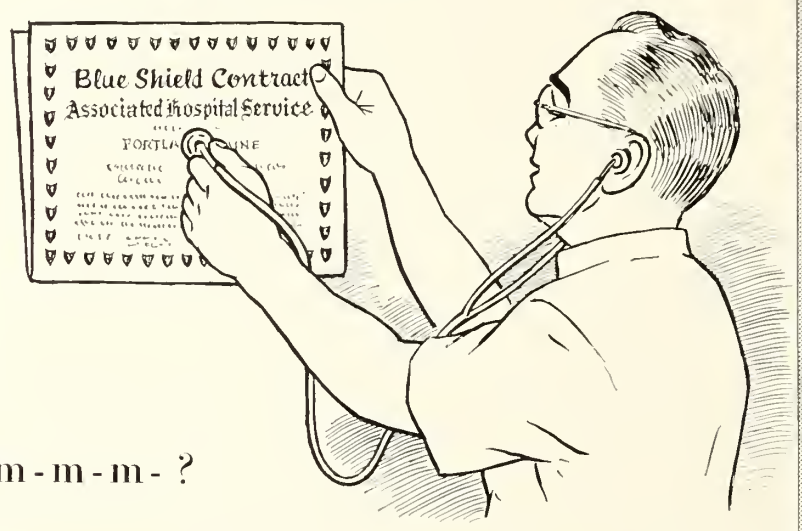
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1. Torosdag, S., Schvartz, N., Fletcher, L., Fertig, H., Schwartz, M. S., Quan, R. F. B., and Bryant, J. M., Pargyline Hydrochloride as an Antihypertensive Agent With and Without A Thiazide, Am. J. Cardiol., 12:822, Dec., 1963.

2. Pollack, P. J., Pargyline Hydrochloride and Methyclothiazide Combined In The Treatment of Hypertension, Cur. Thera. Res., 7:10, Jan., 1965.

3. Bryant, J. M. et al., Antihypertensive Properties of Pargyline Hydrochloride, New Non-Hydrazine Monoamine Oxidase Inhibitor Compared with Sulphonamide Diuretics, J.A.M.A., 178; 406, Oct., 1961.

512214



BP reductions in the recumbent and sitting positions often are nearly as great as in the standing. In clinical trials, the average *recumbent* BP reduction was 36/18 mm. Hg.



The average *standing* reduction in clinical trials was 45/22 mm. Hg. Thus the difference between the standing and recumbent readings was only 9/4 mm. Hg.

Significantly lowers blood pressure in all body positions; less likelihood of orthostatic hypotension

In clinical trials, the average reduction in standing blood pressure was 45/22 mm. Hg.; in the sitting position it was 48/20 mm. Hg.; and in the recumbent position, 36/18 mm. Hg.

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This was reflected in the relatively mild character of side effects seen in clinical trials (see below).

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Onset of antihypertensive action is usually quite smooth. Initial reduction of systolic and diastolic readings is usually seen within a week – maximum reduction in seven to ten days.



Less troublesome side effects may be seen; frequent improvement in "sense of well-being"

Fewer than 1% of patients studied discontinued Eutron therapy because of side effects. This is due in part to the relatively low dosage needed with the combination. Usual recommended dose is one tablet daily—that is, 25 mg. Eutonyl with 5 mg. Enduron. *This is about half the usual therapeutic dose of Eutonyl given alone.* As a consequence side effects may be milder. And, as with Eutonyl given alone, the patient may well note an increased sense of well being. This is in distinct contrast to most other antihypertensive therapy.

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Prescribing information for EUTRON

INDICATIONS: Eutron (pargyline hydrochloride and methyclothiazide) is indicated in the treatment of patients with moderate to severe hypertension, especially those with severe diastolic hypertension. It is not recommended for use in patients with mild or labile hypertension amenable to therapy with sedatives and/or thiazide diuretics alone.

CONTRAINDICATIONS: Eutron is contraindicated in patients with pheochromocytoma, advanced renal disease, paranoid schizophrenia and hyperthyroidism. Until further experience is gained it cannot be recommended for use in patients with malignant hypertension, children (under 12 years of age), or pregnant patients.

The concomitant use of the following is contraindicated: other monoamine oxidase inhibitors; parenteral forms of reserpine or guanethidine; sympathomimetic drugs; foods high in tyramine such as cheese; imipramine and amitriptyline, or similar antidepressants; methyldopa. A drug-free interval of two weeks should separate therapy and use of these agents.

WARNINGS: Pargyline hydrochloride is a monoamine oxidase inhibitor. Patients should be warned against eating cheese, and using alcohol, proprietary drugs or other medication without the knowledge of the physician. When it is necessary to administer alcohol, narcotics (notably meperidine), antihistamines, anesthetics, barbiturates and other hypnotics, sedatives, tranquilizers, or caffeine, these agents can be used cautiously at a dosage of ¼ to ⅕ the usual amount. Avoid parenteral administration where possible. Withdraw pargyline two weeks before elective surgery.

Patients should be warned about the possibility of postural orthostatic hypotension. Those with angina or other evidence of coronary disease should not increase physical activity. Pargyline may lower blood sugar. Potassium depletion is unlikely at the recommended dosage, but if it occurs, adjust dosage or withdraw or provide added natural food sources of potassium; potassium tablets should be avoided wherever possible, as bleeding or obstructive ulceration of the small bowel has been associated with their use; potassium levels should be especially watched if the patient is on digitalis or steroids, or if hepatic coma is impending.

PRECAUTIONS: When determining the antihypertensive effect of Eutron, blood pressure should be measured while the patient is *standing*. Use with caution in hyperactive or hyperexcitable persons. Such persons may show increased restlessness and agitation. Withdraw drug during acute febrile illness. Watch patients with impaired renal function for increasing drug effects or elevation of BUN and other evidence of progressive renal failure; withdraw drug if such alterations persist and progress. Pargyline has not been shown to cause damage to body organs or systems. As with all new drugs, complete blood counts, urinalyses, and liver function tests should be performed periodically. The drug should be used with caution in patients with liver dysfunction. With prolonged therapy, examine patients for change in color perception, visual fields, and fundi.

Elevated blood urea nitrogen, serum uric acid or blood sugar are possibilities attributable to the methyclothiazide in Eutron. Methyclothiazide may also reduce arterial response to pressor amines. Blood dyscrasias, including thrombocytopemia with purpura, agranulocytosis and aplastic anemia, have been seen with thiazide drugs.

SIDE EFFECTS: The use of pargyline may be associated with orthostatic hypotension. Mild constipation, slight edema, dry mouth, sweating, increased appetite, arthralgia, nausea and vomiting, headache, insomnia, difficulty in micturition, nightmares, impotence, delayed ejaculation, rash, and purpura have been encountered with pargyline. Hyperexcitability, increased neuromuscular activity (muscle twitching) and other extra-pyramidal symptoms have been reported. Drug fever is extremely rare. Congestive heart failure has been reported in a few patients with reduced cardiac reserve. Nocturia has been observed with the combination. If side effects persist, despite symptomatic therapy or reduction of the dose, discontinue the drug.

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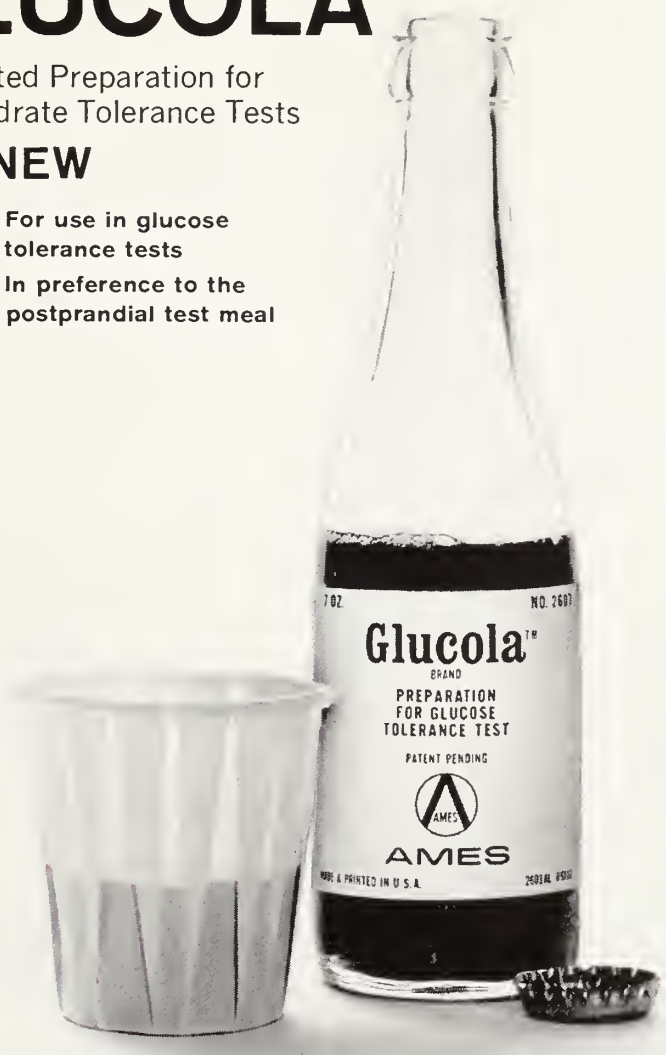
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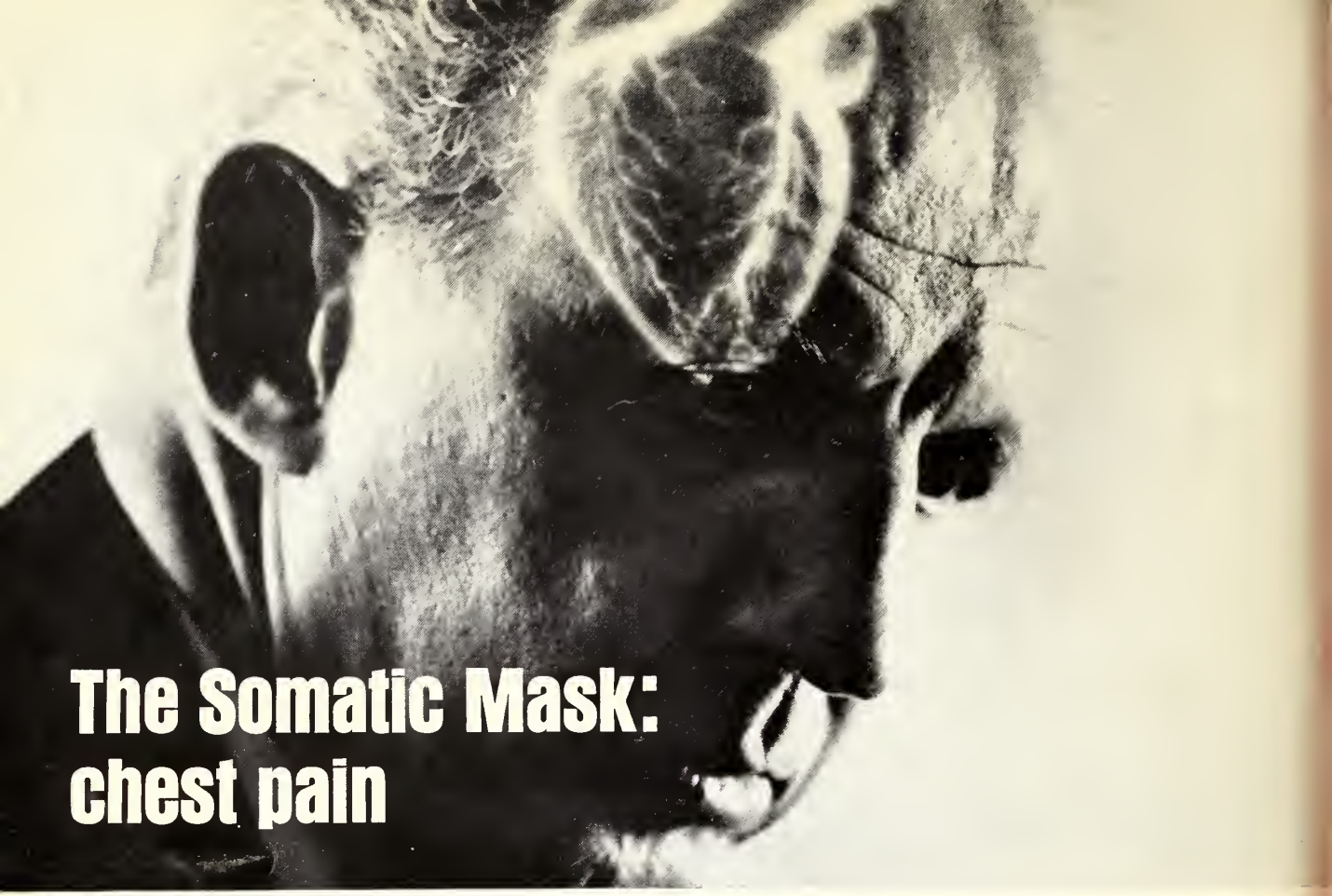
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Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

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1966 Annual Session
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THE JOURNAL
of
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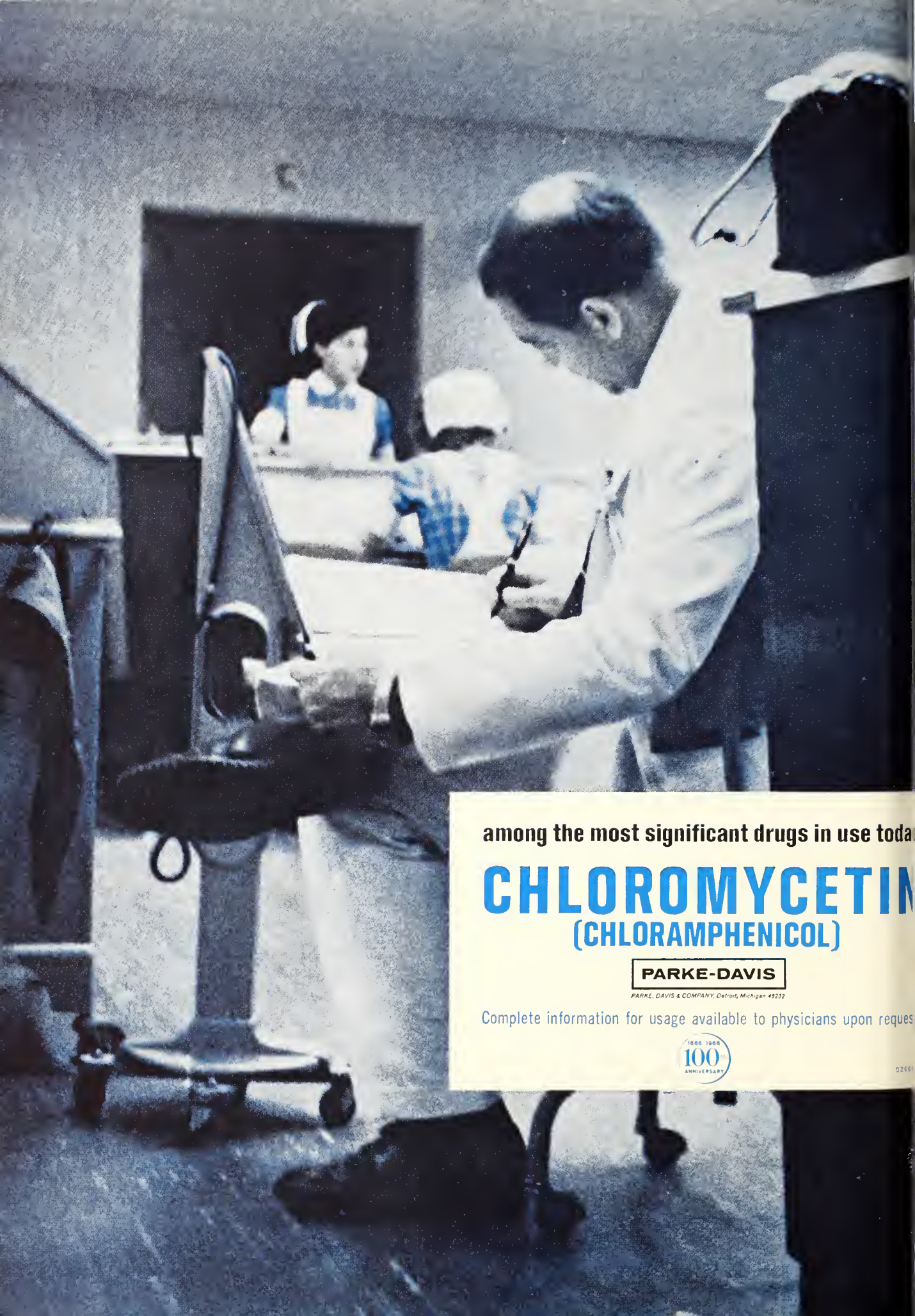
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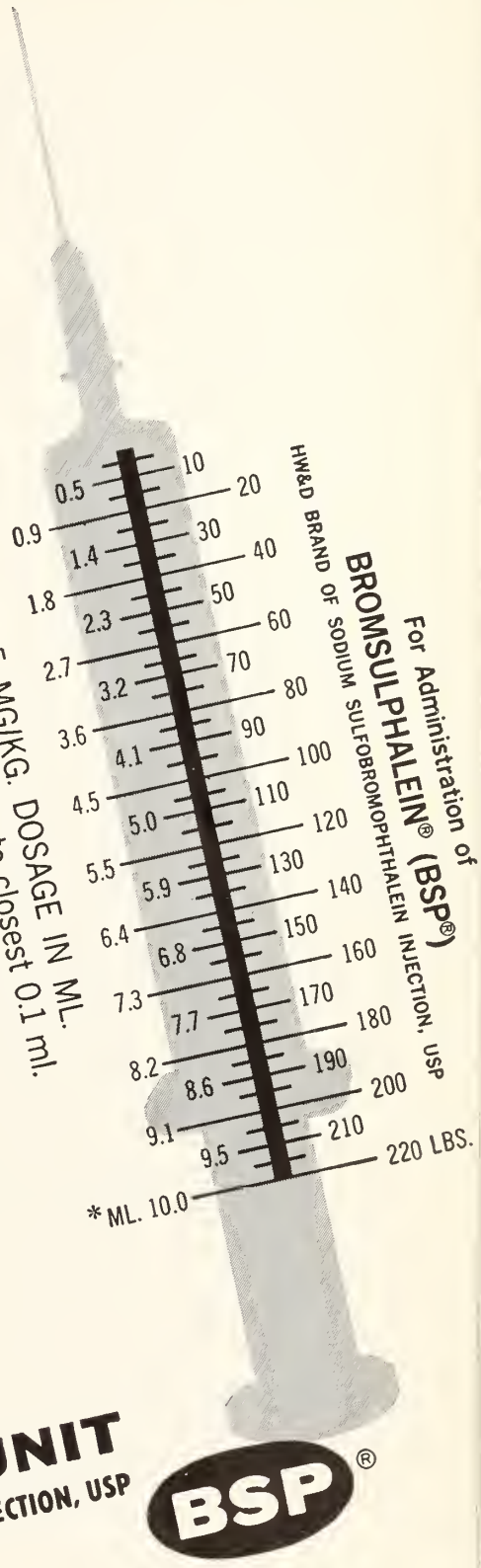
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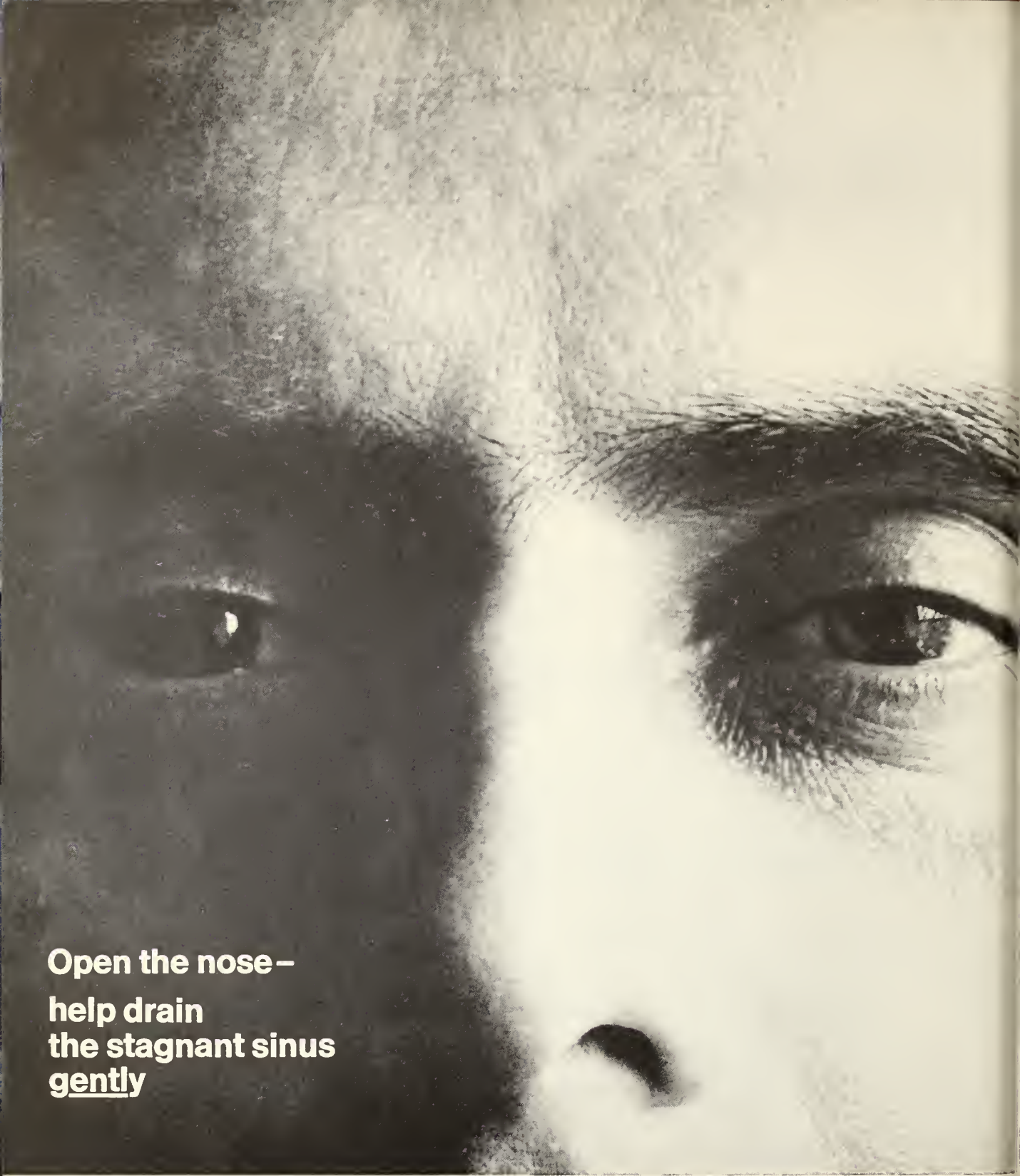
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*Proctor, D. F.: The Nose, Paranasal Sinuses, and Ears in Childhood, Springfield, Ill., Charles C Thomas, 1963, p. 34.

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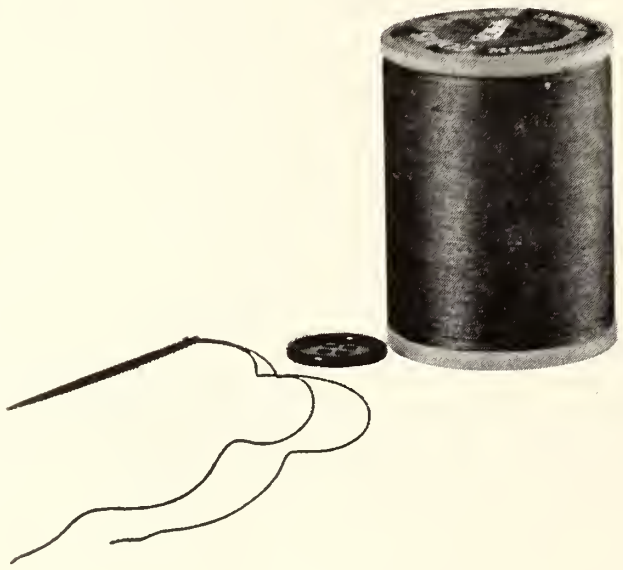
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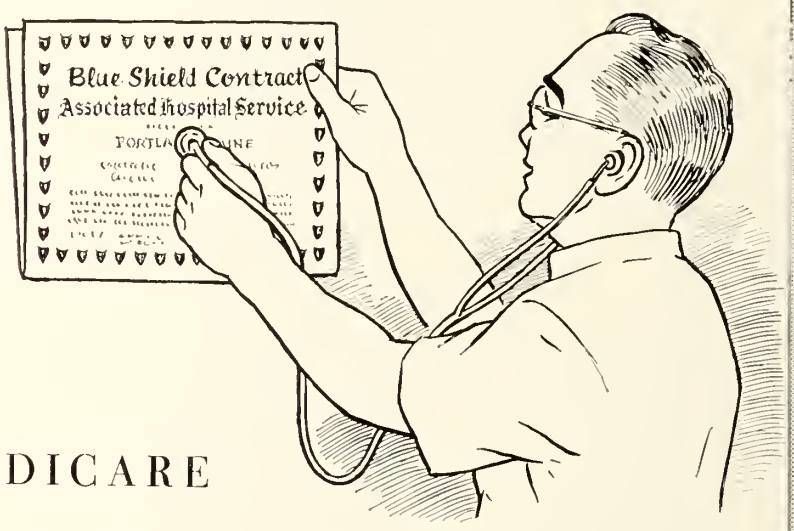
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The Journal
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Maine Medical Association

Volume Fifty-Seven | Brunswick, Maine, March, 1966 | No. 3

Histologically Benign Mixed Tumor of the Parotid Gland with Delayed Metastases*

Waldo A. Clapp, M.D.

By definition, the term "mixed tumor" designates the commonest group of major salivary gland tumors which characteristically show combined traits of epithelial and connective tissue growths. The term "mixed tumor" is the most widely used of the many applied to these neoplasms and does not imply derivation of the tumor from two or more types of tissue. Either epithelial or connective tissue patterns may be dominant or may be in equal balance. Grossly, these tumors vary from 2 to 5 cms. in diameter and are most frequently located within the tail or inferior portions of the parotid gland and present just beneath the lobe of the ear. Sixty percent occur in females. They are well circumscribed usually with a capsule which may be as thick as 1 mm. Secondary cyst formation and hemorrhage are infrequent.

There is considerable controversy as to the histogenesis of so-called mixed tumors of the salivary glands. There are three theories of origin[1]: from connective tissue, from the vascular endothelium of the gland, and from branchial anlage. The glandular epithelial theory appears to be most widely accepted. It seems unlikely that adult connective tissue is capable of epithelial metaplasia; satisfactory evidence[1] can be found of gradual formation of epithelial cells.

The rate of recurrence of mixed tumors following excision is commonly reported to be in the neighborhood of 20-30%.[1] There appears to be likely correlation between histological appearance of the tumors with their tendency to recur. In a review of 877 cases[1] by Foote and Frazell there was only one example of so-called mixed tumor of the parotid gland that was associated with metastasis. Their case was a 26-year-old white female who had a tumor removed from behind the lobe of the right ear in July of 1934. Nine months later a recurrence was removed and a second recurrence was treated with radium in October, 1936. Smaller nodules were surgically excised in 1940, 1943, and 1947. In January, 1953, a routine chest film revealed a spherical density in the lower lobe of the right lung. An exploratory thoracotomy disclosed a 2.5 cm. circumscribed nodule which histologically was identical to the various tumors excised from the parotid gland. This site has remained free from evidence of recurrence for 7 years.

These authors state that, since a metastasis is so rare and totally unpredictable, it should in no way modify current views regarding tumor behavior or conservative clinical management of benign mixed tumors.

Case Report

F.C. is a 40-year-old white male who was admitted to the C.M.G. Hospital 5/18/55 because of a swelling beneath the right ear lobe of 1 year's duration. The mass had increased in size gradually and had not been painful for past 2-3 months. Past history was relevant in that he had rheumatic fever in 1918 with a heart murmur since that time. Physical examination disclosed a firm, discrete nodule at the inferior pole of the parotid gland on the right side measuring 2 cms. in diameter. This was non tender and did not appear fluctuant. There was no palpable regional adenopathy. The remaining physical examination was essentially negative except for a loud systolic murmur heard best over the mitral area. On 5/19 the inferior pole of the gland was resected including the previously described tumor. Pathologically, this tumor was well encapsulated and microscopically the findings were consistent with a benign mixed tumor of the parotid gland. See Figs. 1, 2 and 3.

The patient was next admitted to the C.M.G. Hospital on 12/28/63 with a right hemiplegia, probably embolic, because of the associated auricular fibrillation and rheumatic heart disease.

The third admission was on 2/11/64 because of three nodules: one over the right shoulder, one in the right antero-lateral abdominal wall musculature, and a third nodule in the postero-lateral right thigh of 10 days' duration. These nodules were walnut-size, appeared intra-muscularly, fixed and firm in consistency. All three lesions were excised and were of similar histological nature; namely, "metastatic mixed tumor arising in the mixed tumor of the parotid gland 9 years ago now

*From the Surgical Service, Central Maine General Hospital, Lewiston, Maine.

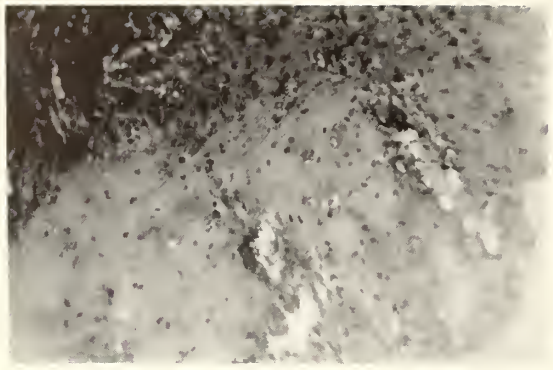


FIG. 1



FIG. 2

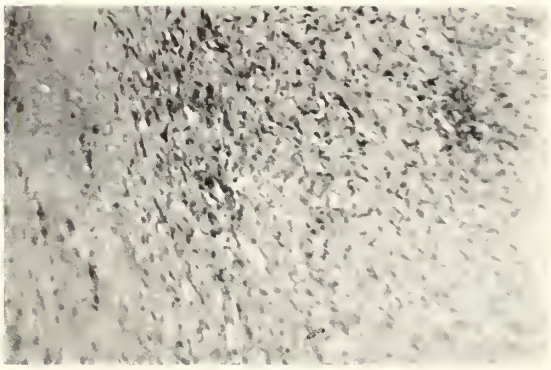


FIG. 3



FIG. 4

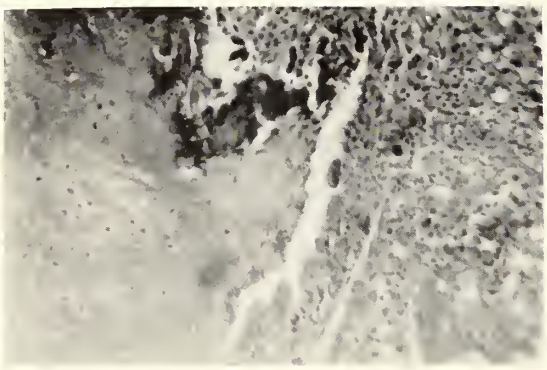


FIG. 5

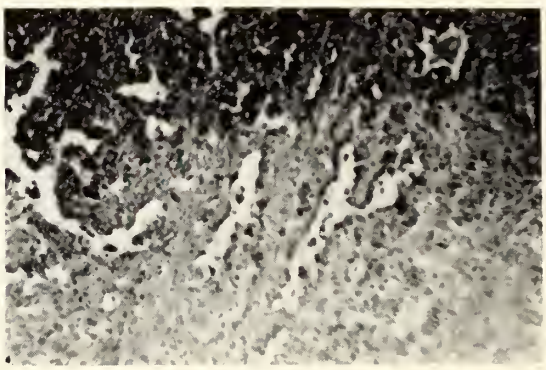


FIG. 6

FIGS. 1, 2, 3. Encapsulated variegated appearance of the parotid tumor removed in 1955.
FIG. 4. Nodule from right chest wall removed in 1964. Note the similarity to FIG. 3.
FIGS. 5, 6. Biopses from right shoulder and right thigh later in 1964. Similar to FIGS. 2 and 3 but with marked anaplasia.

appearing as adenocarcinoma, osteochondrosarcoma and a malignant hemangioma." See Figs. 4, 5 and 6.

The patient was treated with x-ray therapy to the right shoulder for relief of pain; however, he was readmitted 5/18/64 for 5-fluorouracil with some improvement in relief of pain. His final admission on 7/7/64 was for a second course of 5 F.U. which appeared ineffective for recurrent nodules of the right shoulder, right buttocks, and right thigh. Patient expired 9/1/64. Postmortem examination disclosed recurrent metastatic mixed tumors from the parotid to the previously mentioned areas but not to the brain; rheumatic heart disease with a ball valve embolus of the left auricle with emboli to the spleen, kidneys, and brain.

SUMMARY

A case report is presented of a histologically benign mixed tumor of the parotid gland with malignant metastases 9 years following the original excision without evidence of local recurrence.

ACKNOWLEDGMENT

I wish to express grateful acknowledgment to Dr. Charles F. Branch, Pathologist, C.M.G. Hospital, for pathological descriptions and to Dr. Ronald S. Potts, Assoc. Pathologist, C.M.G. Hospital, for photomicrographs and captions.

REFERENCE

1. Armed Forces Institute of Pathology, "Tumors of the Salivary Glands," Frank W. Foote, Jr., M.D., and Edgar L. Frazell, M.D.

215 College Street, Lewiston, Maine

"Misuse of Postprandial Blood Glucose Determinations-Addendum"

RONALD S. POTTS, M.D., C.M.*

In a previous issue,[1] 395 oral glucose tolerances were analyzed and revealed that a large percentage of the abnormal tolerances had two hour true blood glucose levels less than 150 mg.%. Almost all the abnormal tolerances had one hour true blood glucose levels of more than 150 mg.%. This was in support of the theory that two hour postprandial blood glucose determinations were too late to detect glycogenetic insufficiency as a screening procedure; and that one hour postprandial blood glucose levels were more meaningful.

This article is an analysis of 376 additional oral glucose tolerances performed at this hospital during the years 1964 and 1965. Fifty-four percent, or 202, were clearly abnormal by usual criteria.[2] Of these, 176 or 87% had one hour true blood glucose levels of more than 150 mg.%; of these latter, 82 or 41% of all the abnormals had two hour true blood glucose levels less than 150 mg.%. Of all the abnormal tolerances, 106 or 52% had two hour blood glucose levels less than 150 mg.%.

When these time intervals during a glucose tolerance are converted to approximately equivalent postprandial times,[3] it is again seen that the two hour p.c. blood glucose determination is inadequate as a screening procedure in comparison to one hour p.c. determinations.

Table 1 compares the two series and gives the total analysis of the combined series. Fifty-six percent of the total abnormal tolerances yielded normal fasting true blood glucose levels of 65-90 mg.%; 35% of the total abnormal tolerances had both normal fasting levels and two hour levels less than 150 mg.%.

TABLE 1

ANALYSIS OF ORAL GLUCOSE TOLERANCES 1960-1965

	1960-1963	*%*	*1964-1965*	*%*	*totals*	*%*
Total tolerances	395		376		771	
Abnormal tolerances:	218	55	202	54	420	54
1 hr. > 150 mg.%	208	96	176	87	384	91
2 hr. < 150 mg.%			106	52		
1 hr. > 150 mg.%;						
2 hr. < 150 mg.%	87	42	82	41	169	40
normal fasting	124	57	110	55	234	56
normal fasting;						
2 hr. < 150 mg.%	75	35	73	36	148	35

There should now be no doubt that postprandial true blood glucose determinations are necessary to screen for abnormal glycogenesis; and that the commonly designated one-to-two hour postprandial blood glucose determination must be contracted to one hour p.c. determinations to avoid missing fewer abnormal blood glucose levels.

* Associate Pathologist, Central Maine General Hospital, Lewiston, Maine.

REFERENCES

1. Journal Maine Medical Association, March, 1964, Potts, R. S., pg. 42.
2. Miller, S. E., "A Textbook of Clinical Pathology," 6th Edition, pgs. 255-263, Williams & Wilkins Co., Baltimore, 1960.
3. Journal Maine Medical Association, loc. cit.

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Familial Spastic Paralysis*

PHILIP ARCHAMBAULT, M.D.

Familial spastic paralysis is a rare neurological disease of unknown etiology affecting the pyramidal tracts and according to Wechsler,[1] is probably due to a defective anlage. The disease is said to be slowly progressive and confined to the lower extremities. Clinically there is weakness and spasticity of the lower extremities and typically a scissors like gait with equinus deformity of the feet. The signs of this disease consists of hyper-active reflexes, ankle clonus and a positive babinski. Wechsler states that this disease eventually leads to total incapacity.

This is a case presentation of a family in which this disease is prevalent. Those members of the family whom I have observed have shown no evidence of progression of the disease during this short period and no one in this family has died of the disease. The family tree lists 54 individuals, 13 of whom were affected by the disease, 6 males and 7 females.

The first member of this family observed was a 2½ yr. old boy who was first seen in February, 1962. The chief complaint was "walking on his toes" since the age of thirteen months. The onset of walking had been within normal limits. He was a full term first born and the delivery had been entirely normal. On examination he showed a strange gait with equinus of the feet and hyper-extension of the knees on weight bearing. He had hyper-active reflexes, ankle clonus and a positive babinski. The upper extremities were normal as were those of the other patients presented. He was at that time being followed in the crippled children's clinic with the tentative diagnosis of cerebral palsy. In December, 1963 a muscle biopsy of the gastrocnemius muscle was done which was entirely normal. The laboratory work during this admission was entirely normal except for the NPN which was upper limits of normal.

In March, 1963 a twenty-year-old man R.P. was seen because of low back pain of one and a half months duration. This pain had been initiated by pushing a car. This man was remarkable in that he had well developed physique from the waist up but very small and weak legs. He was a weight lifter and was able to lift 170 lbs. I did not know at the time that this was the uncle of R.G. who had been seen a little over a year previously and did not know that they were related until he came to pick up the boy at the hospital after the muscle biopsy in December. This man had stated that he had a nephew with cerebral palsy and not much emphasis was placed on this at the time. The man was admitted to the hospital and treated conservatively and made an uneventful recovery; however, neurosurgical consultation was obtained because of the spastic gait with some crouching, hyper-active reflexes and rather "stiff" hips. A spinal tap produced normal fluid. The neurosurgeon felt that this man might have muscular dystrophy.

In September, 1963 C.G. then age two was seen also because she walked on her toes. She was the sister of R.G. She was also the product of a normal full time pregnancy with normal delivery. She had sat up at six months and walked at fourteen months. Her gait was similar to her brother's but she did not "back knee" as her brother did. She walked with a crouch. The mother of the two children also has an odd gait with only slight spasticity and no real equinus or crouching but hyper-active reflexes and a positive babinski.

The treatment of this disease is the same as that of cerebral palsy. The mother and the uncle of the two children mentioned require no treatment and the uncle is at present employed in a brickyard doing heavy manual labor successfully. In October, 1964 bilateral heel cord lengthening was done on both of the children and there is marked improvement in their gait and they have been able to discard their heavy long leg braces. The heels now come down to the floor well and the gait is much less crouching than it was before.

SUMMARY

A few patients with familial spastic paralysis from a large family affected with this disease has been presented. This disease closely resembles cerebral palsy which is not a familial disease and obtaining a good family history will differentiate between the two. For practical purposes the treatment of this disease is the same as that for cerebral palsy. No progression of the disease has been observed in any member of this family in over 4 years.

REFERENCE

1. Wechsler, Israel S. A Textbook of Clinical Neurology, Sixth Edition, W. B. Saunders Co., 1947, P. 127.

*Presented at Maine Orthopedic Seminar, July 23, 1964.

346 Main Street, Lewiston, Maine

INTERIM MEETING

Maine Medical Association House of Delegates

Sunday, April 3, 1966

THE STOWE HOUSE, BRUNSWICK, MAINE

Dinner at 12:30 P.M. followed by the Business Meeting
Presiding, Linus J. Stitham, M.D., Dover-Foxcroft
Speaker of the House of Delegates

The Order of Business for this meeting, which has been mailed to each of the county delegates and alternates, will include financial statement for 1965, budget proposed for 1967 and other matters to be acted upon at the Annual Meeting in June.

Maine Medical Association Council

The Council will meet at the M.M.A. headquarters in Brunswick, Maine at 10:00 A.M.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Rheumatic Fever and Rheumatic Heart Disease Preventive Services

CHARLES H. OKEY, Ph.D.*

Rheumatic fever and rheumatic heart disease are preventable diseases. Rheumatic fever is preventable in the sense that 10 days of penicillin therapy for every patient having a Group A beta hemolytic streptococcal infection will prevent most attacks of rheumatic fever. Rheumatic heart disease is preventable in the sense that penicillin prophylaxis on a daily basis for those patients with a history of rheumatic fever episode will almost completely eliminate the chances of subsequent streptococcal disease, consequently reducing the probability of further cardiac damage. Means are available to the physicians of Maine to assist them in realizing this potentiality of preventability for their patients. It is the purpose of this presentation to provide the background for this point of view and to describe the facilities available to physicians to accomplish these goals.

Streptococci are part of the normal flora in the pharyngeal tissues of 5% of the population at any given time during the winter months in New England. During pre-epidemic and epidemic periods, the carrier rate may rise to 25%. Generally speaking, only a few of the forty-odd serotypes of the Group A beta hemolytic streptococci are distributed within a population group at any one time. With the introduction of a type not previously found in a group, or the introduction of a type for the first time to an individual, or for reasons not understood but related to the resistance of an individual or a group, streptococcal disease occurs. Symptoms of sore throat with sudden onset and pain on swallowing, headache, fever (incidence is variable) and abdominal pain with nausea and vomiting commonly seen in children are those most commonly reported. Signs of red throat, exudate (particularly after the first day), lymphadenopathy and rash are observed. Some authorities believe the exudate to be a most valuable diagnostic sign. However, all of these symptoms and signs may be produced by agents other than Group A streptococci. Studies based on clinical impressions vs. throat cultures and antibody titers indicate the former to be accurate in only 50% of the cases and in the direction of overdiagnosis. Similarly, studies of rheumatic fever cases seen in centers specializing in this disease, found that from one-half to two-thirds the patients had not been seen by a physician during the antecedent illness. This would seem to indicate, among other things, that the apparently relative mildness of the illness failed to alert the family to the need of medical attention.

Recovery from streptococcal disease results in the development of immunity to the specific streptococcal type involved in the attack. The individual is then protected against further attacks by the same type but is completely susceptible to each of the other types. This explains the frequency of streptococcal diseases in some individuals; each episode is caused by another specific type. Simultaneous with the acquisition of specific immunity, an occasional person develops an additional immunologic factor. This individual, for reasons unknown, becomes hypersensitive to streptococcal antigens. The altered reactivity manifests itself some three weeks later in heart or kidney tissue damage which is designated clinically as rheumatic fever and acute glomerulonephritis. Once the altered reactivity pattern has been established in the rheumatic fever individual, each additional exposure to streptococci precipitates another episode of endocardial mutilation and the crippling process reaches another plateau. Other episodes may follow until the damaged organ is incompatible with life. A mechanism responsible for the damage has not been described. A possible explanation may have been found by Kaplan and Meyerserian[1] who demonstrated that there is an identity between certain Group A streptococcus antigens and heart tissue. Based on this finding, it has been suggested that there may be a mechanism by which the patient's immune reactions result in the formation of autoantibodies to his own heart antigens. If substantiated, this would add yet another to the list of autoimmune diseases in man.

Patients developing a hypersensitivity constitute about 1% of those who have experienced streptococcal dis-

*Director, Public Health Laboratory, Augusta.

ease. The figure varies depending upon the group and its characteristics. Streptococcal outbreaks in young military personnel during World War II resulted in a 3% incidence. This was the highest rate observed for a large group in recent times and probably represents a specialized condition of closely living populations in a particular geographic area. Several authorities have noted the relative mildness of acute rheumatic fever as contrasted with the severe disease observed in the earlier years of this century, as well as a decline in the incidence. Certainly social conditions in this country have influenced the downward trend in rheumatic fever. This belief is based on the observation that the reduced incidence in white populations has not been reflected in the underprivileged non-whites.

Concommitant with recovery, the patient develops streptococcal antibodies against the various antigenic metabolites. One of these, antistreptolysin-O, is used diagnostically in rheumatic fever. Additional tests, based on the same principle but using other metabolites, have been developed but are not widely available because of a lack of standardization.

The American Heart Association, through its state component, the Maine Heart Association, distributes the published recommendations of the Council on Rheumatic Fever and Congenital Heart Disease. These publications, serving as the bases for the recommendations made here, divide rheumatic fever control into two phases: primary prevention and secondary prevention. Primary prevention provides for the early and accurate diagnosis of all Group A streptococcal disease followed by prompt and adequate therapy. Secondary prevention entails long term drug prophylaxis for the individual who has already experienced an attack of acute rheumatic fever.

Throat cultures should be taken on all patients in whom a streptococcal infection is suspected. The cultures can be made by the physician now that prepared plates of sheep blood agar are available commercially, or the specimens on swabs can be submitted to a local laboratory or the Public Health Laboratory for culturing there. Passing the swab over both tonsillar areas and the posterior pharynx will yield the best specimen. In using the Public Health Laboratory outfit, the swab should be allowed to dry for three minutes or so before placing it in the white envelope and the foil envelope. Specimens in the Public Health Laboratory are planted on sheep blood agar plates, screened for beta hemolysis and Group A cultures identified by the fluorescent antibody technic. Reports are mailed out on the day following receipt of the specimen in the laboratory. Telephonic reporting is available if the physician so requests.

It is recommended that all patients yielding Group A streptococci be treated as follows: 600,000 to 900,000 units Permapen® (benzathine penicillin G) administered intramuscularly in children, with the larger dosage given to those over ten years of age, and for adults, 900,000 to 1,200,000 units of the same penicillin. Oral penicillin dosage is set at 200,000 to 250,000 units three or four times a day for a full ten days. The time of ten days is of critical importance in that this period is required to destroy all streptococci. Oral therapy is not as effective in sterilizing the tissues. A follow-up culture should be taken several days after discontinuing oral treatment to determine if the streptococci are present. Sulfonamides are not recommended for the treatment of streptococcal infections. A combination of oral and intramuscular penicillin may be used providing coverage for a full ten days is maintained. For penicillin sensitive patients, Erythromycin is recommended. Tetracycline is not recommended because of the high prevalence of resistant strains of streptococci.

As already mentioned, half or more of the intensively investigated groups of rheumatic fever patients had not been seen by a physician during the antecedent streptococcal infection. Apparently the symptoms were not sufficiently well developed to alarm the patient or the family to the necessity of seeking medical service. The only approach to improving this situation would seem to lie in the direction of patient and family education. Voluntary health agencies should accept this goal as a challenge to their organizations.

The Jones Criteria, used as a guide in the diagnosis of rheumatic fever, were reissued in 1965 in a revised form. Copies of the revised Criteria are available from the Maine Heart Association, 116 State Street, Augusta, Maine. Greater emphasis is placed on laboratory evidence of preceding streptococcal infection. Antistreptolysin-O titers that are elevated or a rising titer in consecutive specimens are the most reliable indications of streptococcal disease capable of producing acute rheumatic fever. A history of isolating Group A streptococci during an antecedent illness or their current presence in the pharynx strengthens the possibility of acute rheumatic fever being present.

Acute rheumatic fever and glomerulonephritis are most often associated with streptococcal nasopharyngitis but there is now convincing evidence that these may also result from impetigo in which Group A streptococci are found. Ten day therapy is indicated for this condition, just as it is for the pharyngeal form.

A number of treatment dosage programs are recommended in rheumatic fever prophylaxis. These choices are described in a publication available from the Maine Heart Association. The Department will furnish penicillin to those rheumatic fever patients under the age of 21 years, seen in the Cardiac Clinics operated by the Division of Maternal and Child Health and who are carried on the register; non-indigent patients pay a nominal amount for the penicillin. The Rheumatic Fever Control Committee of the Maine Heart Association, representing the wholesale and retail druggists of Maine, the medical profession and the Department of Health and Welfare, developed a program in early 1965 to make low cost penicillin available to rheumatic fever

Continued on page 64



normal activity...

promptly...



In children with diarrhea prompt symptomatic control is usually urgently indicated to relieve cramping and to prevent dehydration.

Lomotil halts precipitous progress through the intestines and controls diarrhea with notable promptness, safety and effectiveness.

Experimental evidence[1] has shown that Lomotil is more efficient in this regard than morphine without the latter's manifest disadvantages. In roentgenographic study[2] Lomotil slowed gastrointestinal propulsion within two hours.

At the same time, by diminishing overstimulation of the intestines, Lomotil relieves the abdominal cramps and discomfort so distressing to youngsters.

Lomotil gets children off toast and tea and back to normal diets and normal activity with gratifying celerity.

with LOMOTIL® liquid/tablets

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride .....2.5 mg.
(Warning: may be habit forming)
atropine sulfate .....0.025 mg.

Dosage: For full therapeutic effect—Rx full therapeutic dosage. The recommended initial *daily dosages*, given in divided doses, until diarrhea is controlled, are:

Children:

3 to 6 months—3 mg. (½ tsp* t.i.d.)
6 to 12 months—4 mg. (½ tsp. q.i.d.)
1 to 2 years—5 mg. (½ tsp. 5 times daily)
2 to 5 years—6 mg. (1 tsp. t.i.d.)
5 to 8 years—8 mg. (1 tsp. q.i.d.)
8 to 12 years—10 mg. (1 tsp. 5 times daily)

Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily)

*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one fourth the therapeutic dose.

Precautions: Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is an exempt narcotic preparation of very low addictive potential. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. The subtherapeutic amount of atropine is added to discourage deliberate overdosage.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia.

1. Janssen, P. A. J., and Jageneau, A. H.: A New Series of Potent Analgesics: Dextro 2:2-Diphenyl-3-Methyl-4-Morpholinobutyryl-pyrrolidine and Related Amides. Part 1: Chemical Structure and Pharmacological Activity, J. Pharm. Pharmacol. 9:381-400 (June) 1957.

2. Demeulenaere, L.: Action du R 1132 sur le transit gastro-intestinal, Acta Gastroent. Belg. 21:674-680 (Sept.-Oct.) 1958.

SEARLE

Research in the Service of Medicine

Maine Heart Association Notes

116 State Street, Augusta, Maine

Arcus Senilis and Coronary Artery Disease

"Early observers variously attributed arcus senilis to dryness of the cornea, local vascular impairment, chronic inflammation, or even pressure from the eyelids . . . Detailed histologic observations . . . showed that the lipid of the arcus, present in the membranes and stroma of the cornea, comprises cholesterol, phospholipid, and neutral fat, and the large amounts which may be present, together with the absence of any underlying degenerative change led them to reject the idea that arcus was a simple senile degeneration and to postulate that the fat is derived from the blood and becomes secondarily bound to the corneal tissue.

. . . arcus senilis increases in frequency with advancing years, and it appears earlier in men than in women. It is a frequent accompaniment of pronounced hypercholesterolemia. We consider that its presence or absence in the middle-aged or elderly is of little clinical significance; however, its appearance in men under the age of 40 suggests an alteration in plasma lipids and an increased susceptibility to coronary artery disease."

Submitted by Jacob B. Dana, M.D.

Reference: McAndrew, G.M., Ogston, D., American Heart Journal, Volume 70, pages 838-839, 1965

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Death Rates From Lung Cancer and Other Diseases

Death rates from lung cancer and several other diseases are higher among women cigarette smokers than among women who don't smoke, although total death rates are considerably higher among men than among women, new findings by the American Cancer Society revealed recently.

This is the first time a very large group of women has been traced for the effects of cigarette smoking.

In a paper published by the National Cancer Institute of the U. S. Public Health Service, Dr. E. Cuyler Hammond, director of Statistical Research of the American Cancer Society reported on the impact of smoking-associated diseases on women. Previous reports have dealt with the effects of cigarette smoking on disease and death rates among men.

While citing the higher death rates among men smokers as compared with women smokers the report found that: "Female cigarette smokers (as a group) have been far less exposed to cigarette smoke than have male cigarette smokers of the same ages as judged by number of cigarettes smoked per day, degree of inhalation of the smoke and the number of years they have indulged in the habit."

The study also found that the lung cancer death rate increased among women with the amount of cigarettes smoked, degree of inhalation and also if they started smoking at an earlier age.

Dr. Hammond's report is based on 3,764,571 person-years of experience and 43,221 deaths that occurred among 1,009,229 subjects. This was the number enrolled in the American Cancer Society's huge Cancer Prevention Study. Of these, 440,558 were men and 562,671 were women. They were between the ages of 35 and 84 in the period October 1, 1959 – February 15, 1960 when they were enrolled in this prospective study. All answered detailed questionnaires including information on their smoking habits. More than 68,000 American Cancer Society volunteers conducted the study.

Among both men and women, death rates from the following diseases were much higher in cigarette smokers than in non-smokers: emphysema; cancer of the lung; cancer of the buccal cavity; pharynx, larynx, and esophagus; aortic aneurysm; cancer of the pancreas; and cirrhosis of the liver.

It was found that the death rate from coronary heart disease for men cigarette smokers was 2.81 times as high as for men who never smoked regularly. The coronary heart disease death rate of women cigarette smokers was 2.00 times as high as that of women who never smoked regularly.

In the case of cerebral vascular lesions the pattern was reversed: the death rate of women from this cause was 2.11 times as high for cigarette smokers as compared with non-smokers and in the case of men the death rate from cerebral vascular lesions was 1.50 times higher for smokers than non-smokers.

Death rates from most of the common diseases were higher in men than in women and were far higher in men with a history of cigarette smoking than in women with such a history. This was especially indicated in the rates for lung cancer. The death rate from lung cancer in men (age group 35-84) was 9.20 times higher for cigarette smokers than for those who never smoked regularly. In women (ages 40-74) the death rate for cigarette smokers was 2.20 times as great as those who never smoked regularly. (There were not sufficient cases in the group under 40 to permit an exact comparable selection of women and men.)

However, the tables which separated heavy smokers from light smokers among women, as well as those who did not inhale from those who did, and also those who started smoking younger than 25 from those who began at 25 or older, reveal striking increases of the lung cancer mortality rate. Thus, the new study showed that whereas women who smoked between 1 and 19 cigarettes a day had a lung cancer mortality rate of 1.06 (only slightly higher than those who never smoked regularly), the rate for women who smoked more than 20 cigarettes a day was 4.76 times higher than that of the non-smokers.

For those women who did not inhale, or only inhaled slightly, the death rate from lung cancer was 1.78 times greater than that for non-smokers; for moderate or deep inhalers the rate was 3.70 times greater than for non-smokers.

The new report indicated that among men (for whom data was available) the death rate of ex-cigarette smokers were lower than the death rates of men smoking cigarettes at the time of enrollment in the study. Furthermore, death rates of ex-cigarette smokers decreased with the length of time since they smoked cigarettes. The decline in lung cancer deaths among ex-cigarette smokers parallels the findings of a decrease in the number of cells with atypical nuclei in the bronchial epithelium of ex-cigarette smokers. Similarly with emphysema and coronary disease there is a striking parallel between the statistical association of death rates and cigarette smoking with histologic, cellular and other physiological changes indicative of the development of the diseases.

The new study was published in Monograph #19 of the National Cancer Institute series and is entitled "Epidemiological Study of Cancer and Other Chronic Diseases." It is a memorial to Dr. Harold F. Dorn who was Chief of Biometrics Research Branch, National Institutes of Health of the U. S. Public Health Service; he conducted pioneering studies among veterans on the relation of smoking and health. He died May 9, 1963.

In another paper published in the Dorn Memorial, ACS biometricians explain a phenomenon which tends to show that higher death rates exist among those who have recently given up smoking, compared with those who continue to smoke.

In an analysis of data from the ACS study of more than 1,000,000 persons, Dr. E. Cuyler Hammond and Lawrence Gar-

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Necrologies

Adolphe J. Gingras, M.D.
1896-1966

Adolphe J. Gingras, M.D., 69, of Augusta, Maine died on January 14, 1966. He was born in Rochester, New Hampshire on June 26, 1896, son of Fortunat and Sadie Fortier Gingras.

Dr. Gingras attended Rochester schools, graduated from Laval College and received his medical degree from Laval University School of Medicine in 1923. He did postgraduate work at Tufts-New England Medical Center and at Harvard Medical School. He had practiced medicine in Augusta since 1923 and for many years was a leading obstetrician in that area. He delivered an estimated 8,500 to 9,000 babies in the years around World War II.

Dr. Gingras was a member of the Kennebec County Medical Association, the Maine Medical Association and the American Medical Association. He was also a member of the Knights of Columbus, a long-time member of the Calumet Club and its 13th president in 1933, a member of the board of trustees of the Augusta Water District and the city's Board of Health, and a director of the Depositors Trust Company and the Kennebec Savings Bank. He served in the Medical Corps during World War I.

An operating suite in the new section of the Augusta General Hospital will be named after Dr. Gingras, who was a past president of the medical staff and a former chief of staff.

Surviving are his widow, the former Antoinette Lacombe Gingras; two sons, Adolphe J. Jr., Augusta and Paul J., attending Boston College; two daughters, Mrs. Constance M. Crawford and Mrs. Marie Theresa Ottmann, both of Augusta; three brothers, Alphonse and Isidore J., both of Rochester, N.H. and Dr. Napoleon J. Gingras, Augusta; five sisters, Mrs. Sylvia Gareau, Rochester, N.H.; Sister Lucy of the Sacred Heart and Sister Ida of The Cross, both of Montreal; Sister Rose of the Angels, Rochester, N.H.; and Sister Jeanne Marguerite, Port Haitien, Haiti, all Sisters of the Holy Cross; 16 grandchildren, one uncle, and several nieces and nephews.

Herrick C. Kimball, M.D.
1902-1966

Herrick C. Kimball, M.D., 63, of Fort Fairfield, Maine died on January 3, 1966. He was born in Fort Fairfield, Maine on March 16, 1902, son of Leverett G. and Lizzie F. Savage Kimball.

Dr. Kimball graduated from Fort Fairfield High School in 1918, Bowdoin College in 1922 and received his medical degree from Western Reserve University School of Medicine in Cleveland, Ohio in 1925. He interned and served a residency at the Lakeside Hospital in Cleveland, Ohio and then returned to Fort Fairfield where he practiced medicine for thirty-eight years.

Dr. Kimball was a member of the Aroostook County Medical Society, the Maine Medical Association and the American Medical Association. He was also active in the New England Surgical Society, was a fellow of the American College of Surgeons since 1934, medical advisor to the local Selective Board since 1942, a member of the Advisory Council for Hospital Survey Act and served as medical examiner in Aroostook County from 1946 to 1963. Dr. Kimball was vice-president of the First National Bank, chairman of the board of trustees of the Public Library, director of the Utility District and a member of the Free and Accepted Masons in Fort Fairfield.

Surviving are his widow, Mary Towle Kimball; a daughter, Miss Carolyn Kimball, Boston, Massachusetts; a son, Dr. Philip R. Kimball, Cranston, Rhode Island; a sister, Mrs. Helen Andes, Utica, New York; a half-brother, Eugene, Portland; two grandchildren, a nephew and two nieces.

County Society Notes

CUMBERLAND

One-hundred and fifty members and guests were present at a special meeting of the Cumberland County Medical Society which was held at the Sheraton-Eastland Motor Hotel in Portland, Maine on January 13, 1966.

Richard B. Stephenson, M.D., a member of the society and Research Grants Officer at the National Institutes of Health in Bethesda, Maryland, was the featured speaker. Dr. Stephenson described the development of the Public Law 89-239, the Heart Disease, Cancer, and Stroke Legislation of 1965. He indicated that its purpose is to upgrade medical care through the strengthening of existing medical facilities and explained the regional arrangements, stressing the flexibility of boundaries, and indicating that certain institutions could implement programs dealing with certain aspects of the law while others could specialize in different areas.

Dr. Stephenson stated that the Maine Medical Center would be the only institution in the State of Maine which could qualify as a medical center, although he indicated that other Maine institutions could ally themselves with out-of-state institutions in some aspects of the bill. He frequently repeated that the guide lines were purposely left somewhat flexible so that local groups could use ingenuity in devising unique projects to improve the quality of medical care.

Dr. Stephenson also showed slides and described the buildings and functions of the National Institutes of Health which is obviously the hub of governmental medicine and the organization which will approve, coordinate, and supervise the implementation of the Heart Disease, Cancer, and Stroke Legislation.

ADDENDUM: A meeting was held following the close of the special meeting. A resolution was voted upon to be sent to the City Council of the City of Portland, opposing any unification of the Health and Welfare Departments of the City of Portland.

The Cumberland County Medical Society entertained members of the Woman's Auxiliary at the Sheraton-Eastland Motor Hotel in Portland, Maine on January 20, 1966.

Clement A. Hiebert, M.D. of Portland was the guest speaker for this event. He showed slides and described his experiences in Guinea with the S.S. Hope.

STANLEY B. SYLVESTER, M.D., *Secretary*

LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on January 18, 1966.

Mr. John D. Dow, supervisor of Services to Armed Forces Rejectees for the southern half of the state, outlined the purposes of this program.

The nominating committee consisting of Drs. Samuel L. Belknap, Francis A. Winchenbach and John F. Andrews presented the following slate of officers for 1966 which was approved:

President, Alan W. Zeller, M.D., Damariscotta
Vice-President, Philip O. Gregory, M.D., Boothbay Harbor
Secretary-Treasurer, George W. Bostwick, M.D., Newcastle
Censors, Drs. Samuel L. Belknap, Damariscotta, John F. Dougherty, Bath and William W. Conner, Boothbay Harbor
Program Committee, Drs. Carl R. Griffin, Jr., Boothbay Harbor, Mary J. Tracy, Damariscotta and Charles E. Burden, Bath

William W. Conner, M.D. of Boothbay Harbor gave an illustrated talk on the practice of medicine on an Indian Reservation.

GEORGE W. BOSTWICK, M.D., *Secretary*

KENNEBEC

Thirty-seven members and guests were present at a meeting of the Kennebec County Medical Association held at the Holiday Inn in Augusta, Maine on January 20, 1966.

Richard H. Dennis, M.D. called the meeting to order in the absence of the President, Allan J. Stinchfield, M.D.

Theodore M. Russell, M.D. was the recipient of the prize offered under the new incentive program.

Carl A. Brinkman, M.D. of the Maine Medical Center presented an interesting discussion of the problems and advantages of brain scanning.

LUCIEN F. VEILLEUX, M.D., *Secretary*

PENOBSCOT

The Penobscot County Medical Association has elected the following officers for 1966:

President, Herbert C. Gilman, M.D., Millinocket
President-Elect, Jay K. Osler, M.D., Bangor
Secretary, Charles D. McEvoy, Jr., M.D., Bangor
Treasurer, Thornton W. Merriam, Jr., M.D., Bangor
Delegates to the Maine Medical Association House of Delegates: Drs. George O. Chase, James D. Clement, Jr. and Leonard G. Miragliuolo, all of Bangor, Irvin E. Hamlin, Millinocket and George W. Wood, III, Brewer. Alternates: Drs. John S. Houlihan, Richard A. Gaillard, William M. Shubert, Clement S. Dwyer and Richard V. Duffey, all of Bangor
Executive Council, Drs. Herbert C. Gilman, Chairman, Richard T. Munce, Bangor, Charles D. McEvoy, Jr., George W. Wood, III, Thornton W. Merriam, Jr., Jay K. Osler and Richard V. Duffey
Insurance, Drs. Leonard G. Miragliuolo, Chairman, George W. Wood, III and Clement S. Dwyer
Medical Education and Liaison, Dr. Richard C. Wadsworth, Bangor
Public Relations, Drs. Frederick C. Emery, Bangor, Chairman and James D. Clement, Jr.
Program Committee, Drs. George O. Chase, Chairman, Edward J. Hughes, Jr., Byron V. Whitney, Benjamin L. Shapero and Eugene E. Brown, all of Bangor
Diabetes Committee, Drs. Harold D. Cross, Chairman and John C. Bjorn, both of Hampden Highlands

CHARLES D. MCEVOY, JR., M.D., *Secretary*

ANDROSCOGGIN

At the annual meeting of the Androscoggin County Medical Association the following officers were elected for 1966:

President, Harvey J. Proulx, M.D., Lewiston
Vice-President, Daniel R. Shields, M.D., Lewiston
Secretary-Treasurer, Donald L. Anderson, M.D., Lewiston
Delegates to the Maine Medical Association House of Delegates: Drs. Henry C. Thacher, Auburn, Edward L. Reeves, Morris E. Goldman and Joseph J. Rando, all of Lewiston. Alternates: Drs. Philip L. Archambault, Lewiston, Frederick B. Lidstone and Charles A. Hannigan, both of Auburn, and Harvey J. Proulx
Board of Censors, Drs. Daniel A. Rock, Louis N. Fishman and Reuben Leitman, all of Lewiston.

DONALD L. ANDERSON, M.D., *Secretary*

PISCATAQUIS

The following officers were elected for 1966 at the annual meeting of the Piscataquis County Medical Society:

President, Norman H. Nickerson, M.D., Greenville
Vice-President, Felix M. Garcia-Rey, M.D., Milo
Secretary-Treasurer, Isaac Nelson, M.D., Greenville
Delegate to the Maine Medical Association House of Delegates: Dr. Linus J. Stitham, Dover-Foxcroft. Alternate: Dr. Charles H. Lightbody, Guilford

ISAAC NELSON, M.D., *Secretary*

WASHINGTON

The following members of the Washington County Medical Society have been elected to serve as delegate and alternate to the Maine Medical Association House of Delegates:

Delegate: Dr. Hazen C. Mitchell, Calais. Alternate: Dr. James C. Bates, Eastport

KARL V. LARSON, M.D., *Secretary*

AMERICAN CANCER SOCIETY–Maine Division, Inc. — *Continued from Page 59*

finkel indicate that the "former smoker" group is actually weighted with people in poor health and in many instances they became ex-smokers because of symptoms which influenced them to stop smoking.

The statistical "artifact" occurs only among those who have stopped smoking for one year or less. Former smokers who have been off cigarettes for more than a year show a steady improvement in death rates, eventually approaching the "never smoked" category after enough years have passed.

Another factor which influences the higher death rates among first-year ex-smokers, according to the authors, is the observation that a substantial number returned to smoking cigarettes after being included as ex-smokers when initially enlisted in the study.

Adjusting these two factors in their data – current or recent ill health, and a return to cigarette smoking – Hammond and Garfinkel found that even for first-year non-smokers death rates were lower than for current cigarette smokers, and their death rates continued to decline rapidly over the years.

From American Cancer Society News Service Release

News, Notes and Announcements

Dr. MacVane Appointed Director of Medical Education

William L. MacVane, Jr., M.D., has been appointed director of medical education at Mercy Hospital.

Dr. MacVane will continue his practice of general and thoracic surgery, using both the Mercy Hospital and Maine Medical Center.

Dr. MacVane was graduated from Williams College, attended Johns Hopkins Medical School and served his internship at Union Memorial Hospital, Baltimore, Maryland.



He served residencies at the Maine General Hospital and the Veterans Administration Hospital at Togus, and a residency in thoracic surgery at the VA Hospital in Oteen, North Carolina.

From 1943 to 1946, he was a medical officer in the U.S. Army's 61st Portable Surgical Hospital, serving in New Guinea, the Philippines and Japan and reached the rank of major.

He is a fellow in the American College of Surgeons, counselor for Maine of the American Thoracic Society, governor for Maine of the American College of Chest Physicians and a member of the New England Surgical Society, the American Board of Surgery, the American Board of Thoracic Surgery, the Maine Medical Association and the Cumberland County Medical Association.

State of Maine Board of Registration of Medicine
Secretary — George E. Sullivan, M.D.
Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine November 9-11, 1965

Through Examination

Abdalla Y. Abdalla, M.D., 19 Bedford St., Bath, Me.
Corrado M. Baglio, M.D., 238 N. Dithridge St., Pittsburgh, Pa.
Jorge Caceres, M.D., 327 West 30 St., New York, N.Y.
Haldane D. Clarke, M.D., 2609 Loftus Ave., Oceanside, N.Y.
Johan M. Cornelis, M.D., Royal Victoria Hosp., Montreal, Can.
Delia M. C. Montes Gallo, M.D., Hanna Pavilion, University Hospitals, Cleveland, Ohio
David I. Gorescu, M.D., 1492 Montgomery Ave., c/o Rigler, Bronx, N.Y.
Henri R. Hardy, M.D., Louise Obici Mem. Hosp., Suffolk, Va.
Nicola Y. Katf, M.D., 2200 McCoy St., Kansas City, Mo.
Paul R. Levesque, M.D., 47 Westchester Rd., Newton, Mass.
Mario H. Melamedoff, M.D., 141 Monhagen Ave., Middletown, N.Y.
Ahmet S. Satir, M.D., Augusta State Hosp., Augusta, Me.

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Alice W. Dewdney, M.D., 1419 Topeka, Topeka, Kans.
Hal M. Freeman, M.D., Retina Foundation, Boston, Mass.
Alexander R. Gaudio, M.D., 160 Stillwater Ave., Bangor, Me.
Irwin M. Pasternak, M.D., Community Gen. Hosp., Fort Fairfield, Me.
Philip C. Richardson, M.D., 2155 Cram Pl., Ann Arbor, Mich.
Richard A. Rudders, M.D., U.S. Naval Radio Sta., Cutler G, E. Machias, Me.
Nancy H. Stewart, M.D., Flatrock Rd., Fitchburg, Mass.
Dudley B. Tyson, M.D., Bangor, Me.

New England Postgraduate Assembly Meeting Re-scheduled

The November, 1966, New England Postgraduate Assembly, sponsored by the Council of the New England State Medical Societies, has been re-scheduled to:

JANUARY 17, 18, 19, 1967
Statler Hilton Hotel
Boston, Massachusetts

Reason: So it will not conflict with the American Academy of General Practice annual meeting being held in Boston in mid-October 1966.

William H. Baker, M.D., Massachusetts General Hospital, 275 Charles Street, Boston, Massachusetts, is chairman of the Program Committee.

Book Review

Fracture Problems. By William H. Harris, M.D., William N. Jones, M.D. and Otto E. Aufranc, M.D. Published by The C. V. Mosby Company, St. Louis, 1965. 371 pages with illustrations. Price $20.00.

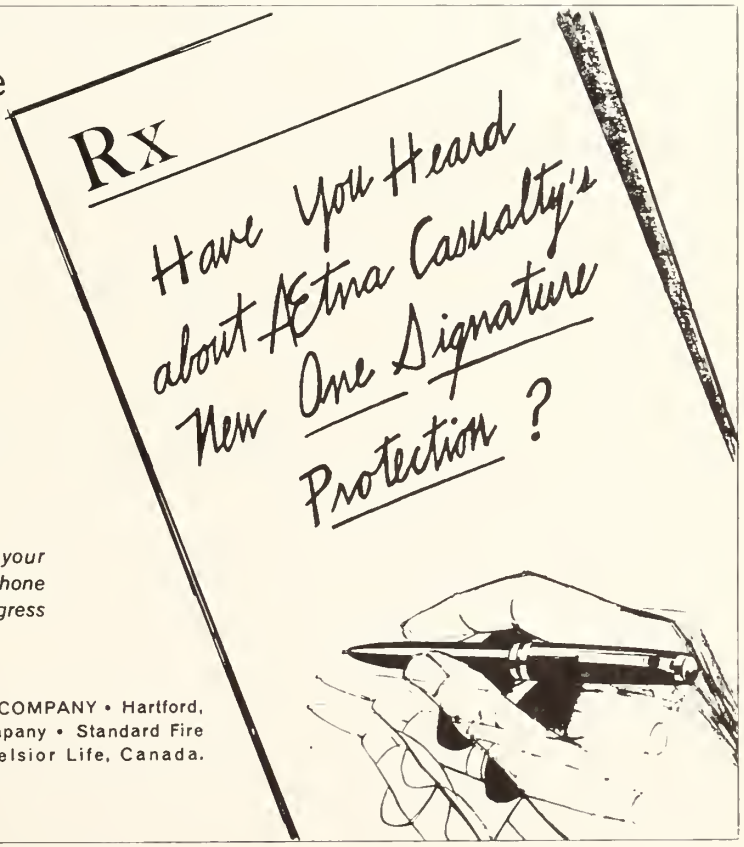
This book represents a compact evaluation of numerous severe fracture problems involving many different portions of the body and also a brief section on pathologic fractures and the management of these. Essentially, it is numerous case presentations with various opinions expressed by different authors as to their method of handling and also concensus impressions by other members of the panel that were given as the case was presented. Some very excellent and complicated cases are presented along with a conservative approach when indicated, and an operative approach when this was the more conservative method of therapy. A book well worth reading and reviewing for anyone who is involved in the treatment of fractures, not only the orthopedic surgeon, but also the general surgeon and general physician to serve as a guide line for the management of some rather severe fractures. Things that can and cannot be accomplished and some cardinal principles of fracture management are well presented, not only from the point of view of the management of the fracture but of the entire patient who suffers multiple severe injuries. It emphasizes the necessity for sometimes ignoring the fracture temporarily, and treating the more serious complications and general medical problems that the patient may have, and then treating the fracture when the patient's condition will allow it. In general, this is an excellent book with very straightforward discussion of some of the problems involved.

JAMES L. FIFE, M.D., Brunswick, Maine

To members of the Maine Medical Association . . .

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- *Office Premises Liability Insurance* for claims arising from the operation of your office
- *"All Risks" Insurance* for loss of or damage to your professional equipment

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DEPT. OF HEALTH & WELFARE—*Continued from page 55*

patients through their physicians. Approximately 200 patients are now enrolled in the program. A physician may enroll the patient on forms provided by the Maine Heart Association. Four prescription blanks, each calling for one hundred 200,000 units of penicillin tablets are sent to the physician. The prescriptions are given to the patient quarterly and the charge for the supply is $2.00.

REFERENCE
1. Kaplan, M. H. and Meyerserian, M. Lancet, I:760, 1962.

Letter to the Editor

Eighty-eight years ago, Nat Baily Twycross Barker was born in Dresden, Maine. Educated at Bowdoin College and Maine Medical School with postgraduate work in New York City, Nat Barker became one of that early line of beloved country doctors who were among the truly "greats" of this country in medicine.

He began his practice in Woodland, Maine on the old St. Croix, sixty-one years ago. During his twenty-three years there, he attended over twenty thousand accident cases. One day when construction work was heavy he had twenty-seven accidents. In a threatened smallpox epidemic he vaccinated ninety-six percent of all the children, big and little. During his lifetime, Dr. Barker has delivered close to twenty-five hundred babies.

Naturally compassionate, with a keen feeling for the ills of mankind, Dr. Nat became guide, counselor and friend to his patients, old and young, rich or poor. People who lived in primitive tar papered shacks and brush huts were cheered and comforted by his presence. From babyhood to old age, they had one thing in common—their love and admiration for their doctor.

On the coldest, snowiest nights he has gone out with horse and sleigh, a shovel, and snowshoes far into the country to care for the isolated needy, the sick and the dying. Through the years he has given away tons of books, magazines and clothing, along with food and medicine, but most important of all he has given of himself, his medical wisdom, his sound good judgment, and his unequaled kindness of heart. Medicine in Maine will be a much poorer thing when Dr. Nat Barker is no longer a part of it. . . .

In World War II, he examined around eight hundred men for the armed services and holds the honorary gold medal for service given by the President.

He has received the fifty-year pin, the fifty-five year pin and the sixty-year pin given by the Maine Medical Association.

The Barker school in Woodland, Maine is named for him, and five babies—four boys and one girl. During much needed vacations with his wife, Cathy, a former school teacher and a highly intelligent and adventurous traveler, he has traveled the world over. During these periods twenty-five men had their schooling in country doctor practice.

These dedicated men like Dr. Nat Barker, come closest to the hearts of their patients for they are present at all hours; to hold back the grim spector of death, to give greater health to a loved one, to bring new life into the world, and to give comfort when life must end. . . .

Like the greatest of all physicians, Dr. Barker has spent his life going about doing good. One day he will hear the kindest of all voices, a heavenly voice say, "Well done Thou good and faithful servant, enter thou into thy reward."

ADRIAN H. SCOLTEN, M.D., Portland, Maine

DEPROL®
meprobamate 400 mg. +
benactyzine hydrochloride 1 mg.

Indications: 'Deprol' is useful in the management of depression, both acute (reactive) and chronic. It is particularly useful in the less severe depressions and where the depression is accompanied by anxiety, insomnia, agitation, or rumination. It is also useful for management of depression and associated anxiety accompanying or related to organic illnesses.

Contraindications: Benactyzine hydrochloride is contraindicated in glaucoma. Previous allergic or idiosyncratic reactions to meprobamate contraindicate subsequent use.

Precautions: *Meprobamate*—Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of preexisting symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage.

Benactyzine hydrochloride—Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria.

Meprobamate—Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Dosage: Usual starting dose, one tablet three or four times daily. May be increased gradually to six tablets daily and gradually reduced to maintenance levels upon establishment of relief. Doses above six tablets daily are not recommended even though higher doses have been used by some clinicians to control depression and in chronic psychotic patients.

Supplied: Light-pink, scored tablets, each containing meprobamate 400 mg. and benactyzine hydrochloride 1 mg.
Before prescribing, consult package circular.

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Effective in a wide range of everyday infections —respiratory, urinary tract and others—in the young and aged—the acutely or chronically ill—when the offending organisms are tetracycline-sensitive.

Warning — In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of discomfort.

Precautions and Side Effects — Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncracy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg of demethylchlortetracycline HCl.

Tablets: film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

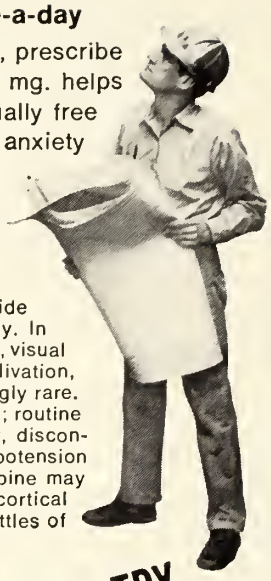
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Lederle

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Blueprint for dealing with tension due to stress — Prolixin — once-a-day

For the patient who must be on the job mentally as well as physically, prescribe Prolixin. The prolonged tranquilizing action of as little as one or two mg. helps him cope with tension all day long. Markedly low in toxicity and virtually free from usual sedative effects, Prolixin is effective in controlling both anxiety associated with somatic disorders and anxiety due to environmental or emotional stress. Patient acceptance is good — because Prolixin is low in cost, low in dosage and low in sedative activity. Prescribe Prolixin.



Side Effects, Precautions, Contraindications: As used for anxiety and tension, side effects are unlikely. Reversible extrapyramidal reactions may develop occasionally. In higher doses for psychotic disorders, patients may experience excessive drowsiness, visual blurring, dizziness, insomnia (rare), allergic skin reactions, nausea, anorexia, salivation, edema, perspiration, dry mouth, polyuria, hypotension. Jaundice has been exceedingly rare. Photosensitivity has not been reported. Blood dyscrasias occur with phenothiazines; routine blood counts are recommended. If symptoms of upper respiratory infection occur, discontinue the drug and institute appropriate treatment. Do not use epinephrine for hypotension which may appear in patients on large doses undergoing surgery. Effects of atropine may be potentiated. Do not use with high doses of hypnotics or in patients with subcortical brain damage. Use cautiously in convulsive disorders. **Available:** 1 mg. tablets. Bottles of 50 and 500. For full information, see Product Brief.

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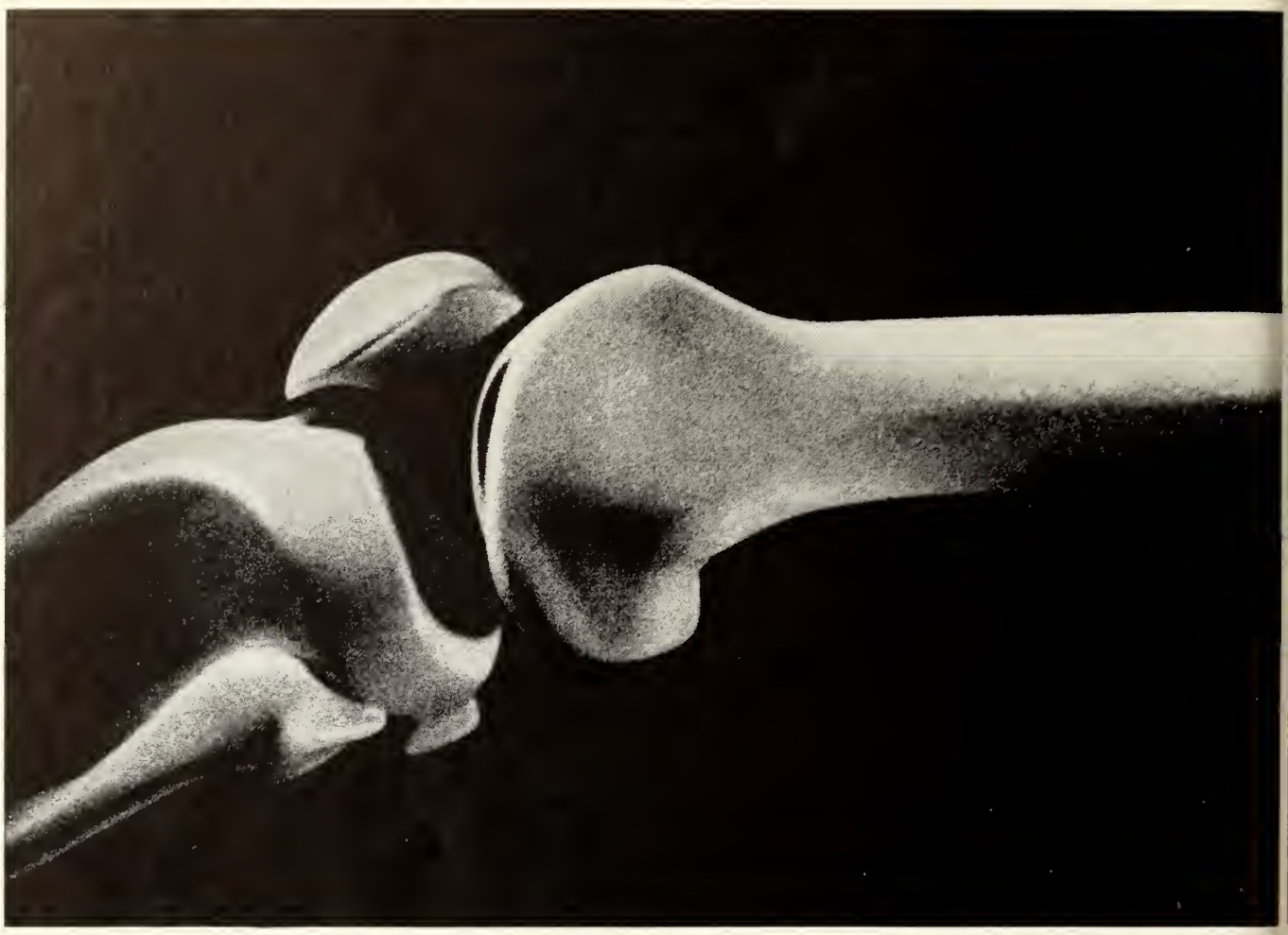
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Contraindications
Edema, danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile, or when other potent drugs are given concurrently. Large doses are contraindicated in patients with glaucoma.

Precautions
Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should be closely supervised and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Make regular blood counts. Use greater care in the elderly.

Warning
If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy.

Adverse Reactions
The most common are nausea, edema and drug rash. Hemodilution may cause moderate fall in red cell count. The drug may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, generalized allergic reaction, stomatitis, salivary gland enlargement, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hepatitis, jaundice, and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently.

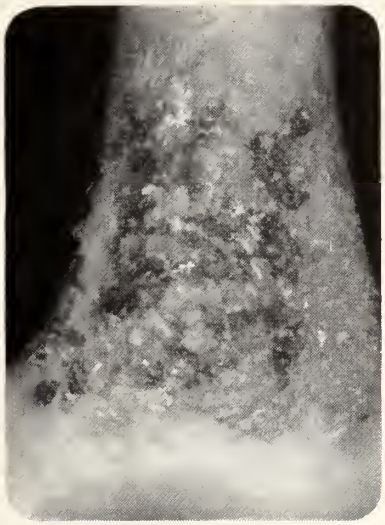
Dosage
The initial daily dosage in adults is 300-600 mg. daily in divided doses. In most instances, 400 mg. daily is sufficient. When improvement occurs, dosage should be decreased to the minimum effective level: this should not exceed 400 mg. daily, and is often achieved with only 100-200 mg. daily.

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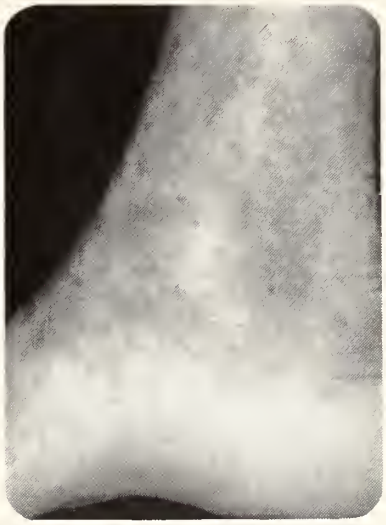
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eczema: scourge of childhood



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After treatment—with ARISTOCORT Topical Ointment 0.1% for two weeks

RISTOCORT® Triamcinolone Acetonide Topicals have oved exceptionally effective in the control of various rms of childhood eczema: allergic, atopic, nummular, oriatic, and mycotic.

In most cases responsive to topical ARISTOCORT, e 0.1% concentration is sufficiently potent. The 0.5% ncentration provides enhanced topical activity for tients requiring additional potency for proper relief.

dministration and Dosage: Apply sparingly to the affected ea 3 or 4 times daily. Some cases of psoriasis may be more ectively treated if the 0.1% Cream or Ointment is applied der an occlusive dressing.

ntraindications: Tuberculosis of the skin, herpes simplex, icken pox and vaccinia.

ecautions and Side Effects: Do not use in the eyes or in ear (if drum is perforated). A few individuals react unorably under certain conditions. If side effects are enuntered, the drug should be discontinued and appropriate measures taken. Use on infected areas should be attended with caution and observation, bearing in mind the potential spreading of infection and the advisability of discontinuing therapy and/or initiating antibacterial measures. Generalized dermatological conditions may require systemic corticosteroid therapy. Steroid therapy, although responsible for remissions of dermatoses, especially of allergic origin cannot be expected to prevent recurrence. The use over extensive body areas, with or without occlusive nonpermeable dressings, may result in systemic absorption. Appropriate precautions should be taken. When occlusive nonpermeable dressings are used, miliaria, folliculitis and pyodermas will sometimes develop. Localized atrophy and striae have been reported with the use of steroids by the occlusive technique. When occlusive nonpermeable dressings are used, the physician should be aware of the hazards of suffocation and flammability. The safety of use on pregnant patients has not been firmly established. Thus, do not use in large amounts or for long periods of time on pregnant patients.

Packages: Tubes of 5 Gm. and 15 Gm.; ½ lb. jar.

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Miltown® (meprobamate)

Indications: 'Miltown' (meprobamate) is effective in relief of anxiety and tension states. Also as adjunctive therapy when anxiety may be a causative or otherwise disturbing factor. Although not a hypnotic, 'Miltown' fosters normal sleep through both its anti-anxiety and muscle-relaxant properties.

Contraindications: Previous allergic or idiosyncratic reactions to meprobamate or meprobamate-containing drugs.

Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

Supplied: In two strengths: 400 mg. scored tablets and 200 mg. coated tablets.

Before prescribing, consult package circular.

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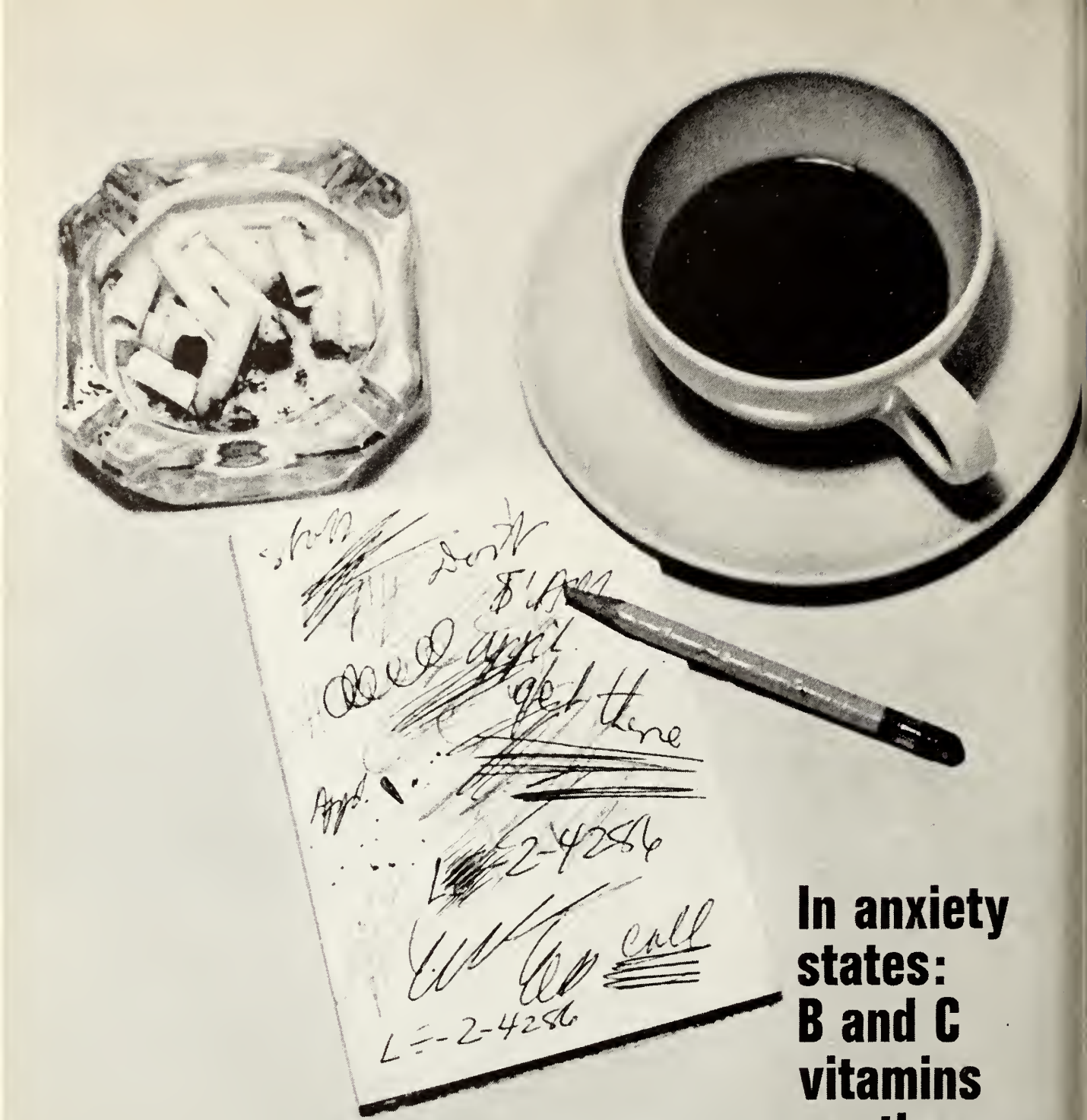
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Stress formula vitamins are an important supportive measure in maintaining the nutritional status of the emotionally disturbed patient. With STRESSCAPS, B and C vitamins are present in therapeutic amounts to meet increased metabolic demands. Patients with anxiety, and many others undergoing physiologic stress, may benefit from vitamin therapy with STRESSCAPS.

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Vitamin B_{12} Crystalline	4 mcgm
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Recommended intake: Adults, 1 capsule daily, for the treatment of vitamin deficiencies. Supplied in decorative "reminder" jars of 30 and 100; bottles of 500.

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With Librium (chlordiazepoxide HCl), the weight of a five-year record of efficacy and safety in clinical use is supported by over 630 reports in the medical literature. Its virtually specific antianxiety action normally reduces disturbing emotional complaints promptly without compromising the patient's mental alertness or ability to perform normal functions. Decisive results are often seen in patients who had not improved on previously used psychotropic drugs.

In prescribing: Dosage—Adults: Mild to moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d.; severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d.

Side Effects: Side effects, usually dose-related, include drowsiness, ataxia, minor skin rashes, edema, menstrual irregularities, nausea and constipation. When treatment is protracted, blood counts and liver function tests are advisable. Paradoxical reactions may occasionally occur in psychiatric patients. Individual maintenance dosages should be determined.

Precautions: Advise patients against possibly hazardous procedures until maintenance dosage is established. Though compatible with most drugs, use care in combining with other psychotropics, particularly MAO inhibitors or phenothiazines; warn patients of possible combined effects with alcohol. Observe usual precautions in impaired renal or hepatic function, in long-term treatment and in presence of depression or suicidal tendencies. Exercise caution in administering drug to addiction-prone patients or those who might increase dosage; withdrawal symptoms, similar to those seen with barbiturates or meprobamate, can occur upon abrupt cessation after prolonged overdosage. Caution should be exercised in prescribing any therapeutic agent for pregnant patients.

Supplied: Capsules, 5 mg, 10 mg and 25 mg, bottles of 50.

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(chlordiazepoxide HCl)

Librium is indicated whenever anxiety is part of the clinical profile, such as in...



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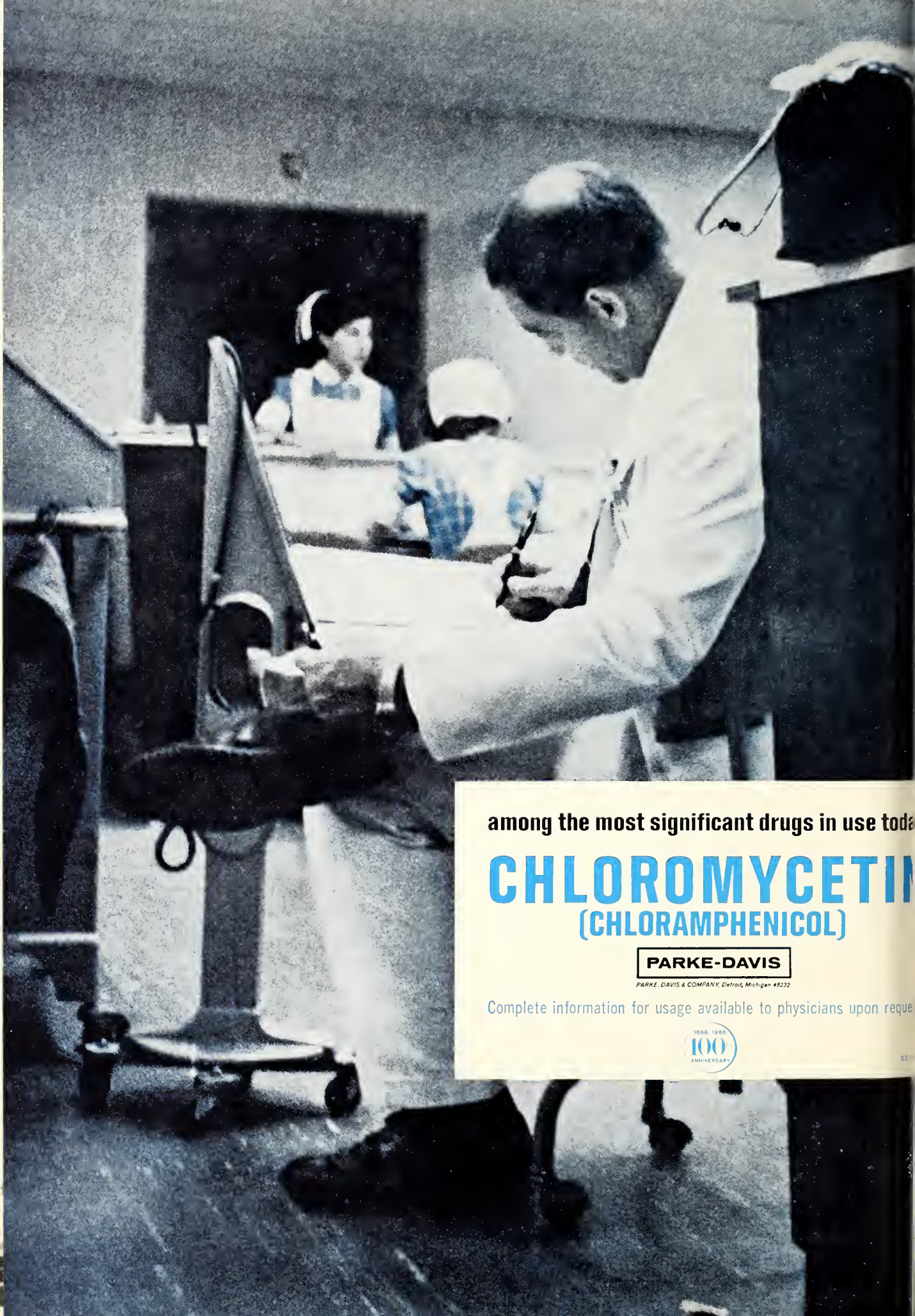
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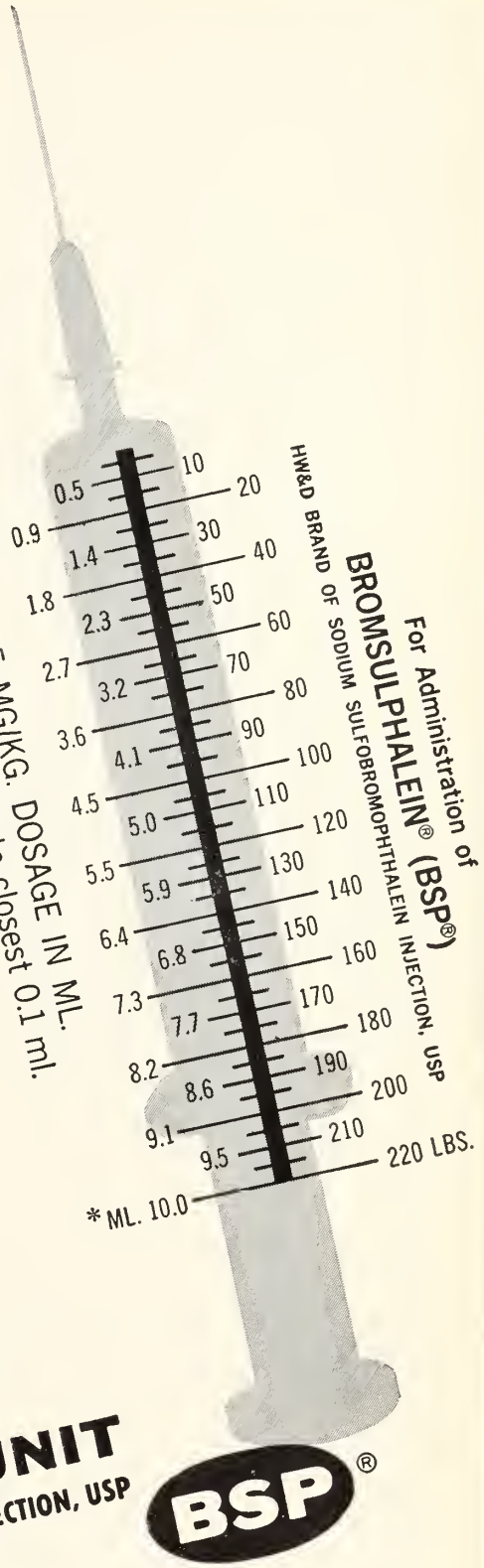
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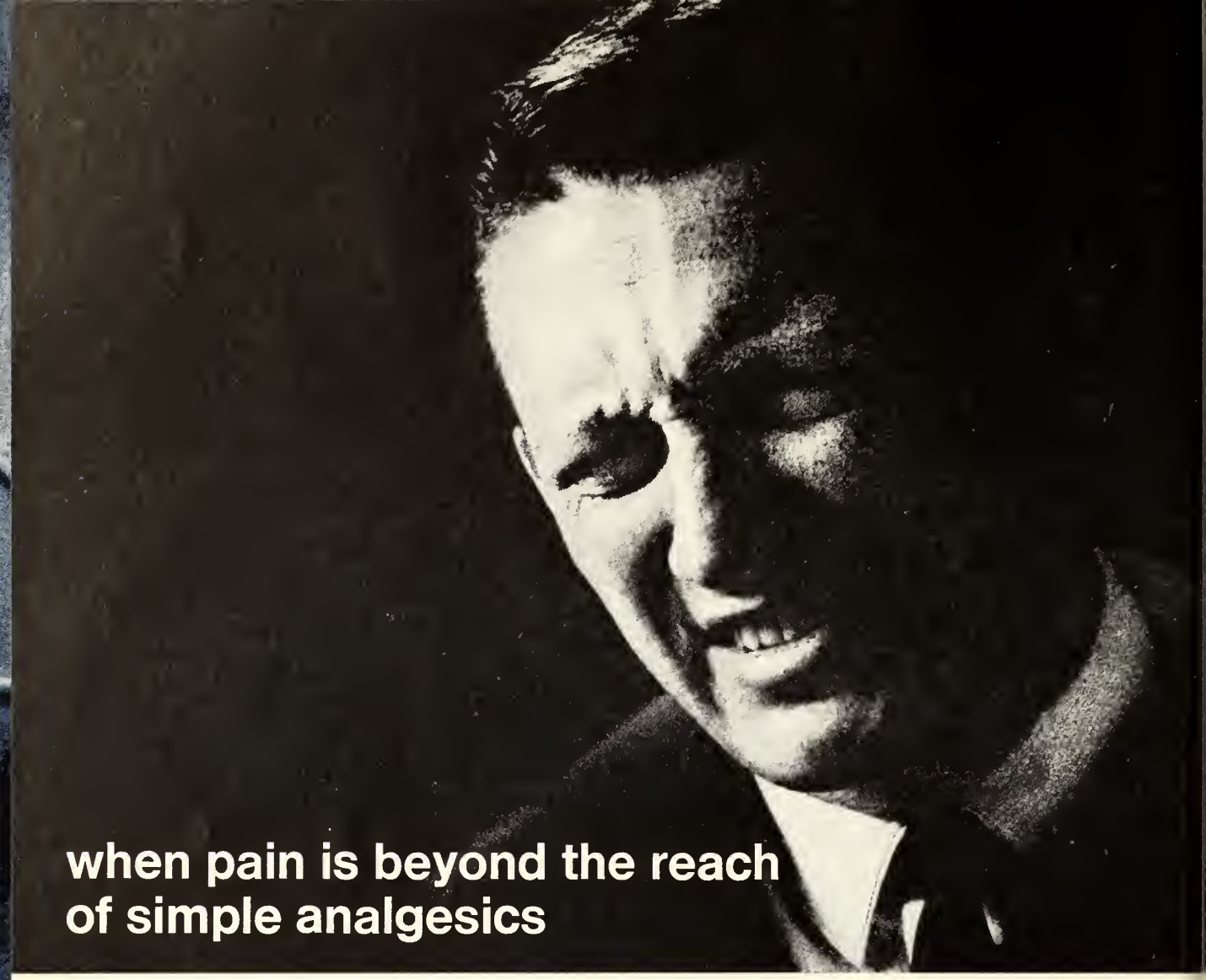
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1. Collective studies, Department of Medical Research, Winthrop Laboratories.

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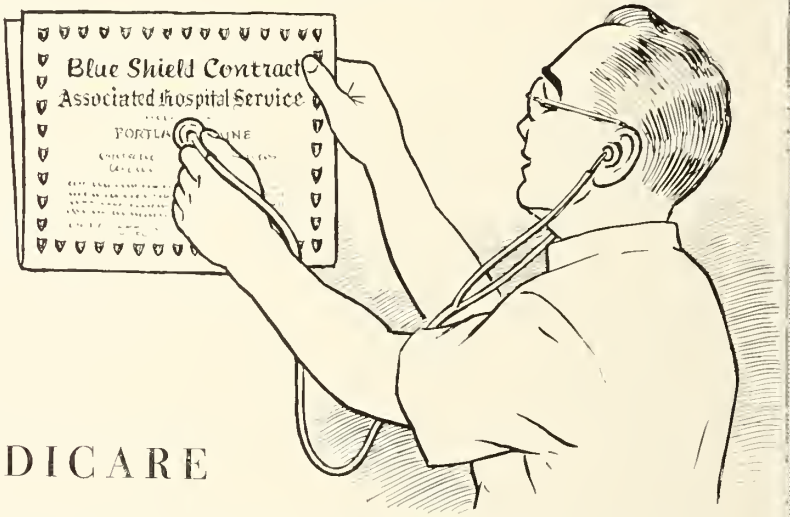
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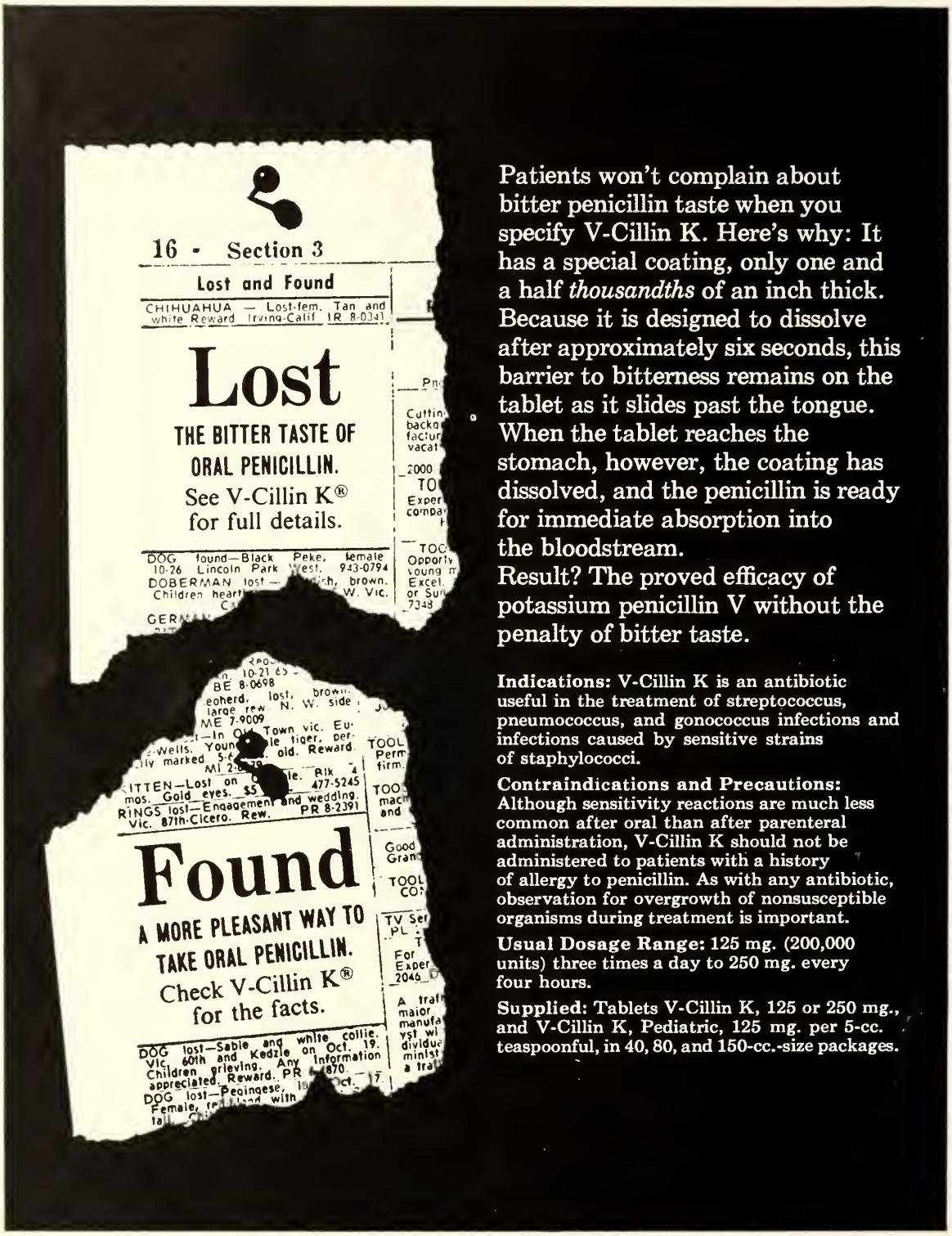
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Volume Fifty-Seven | Brunswick, Maine, April, 1966 | No. 4

Boston Medical Reports

A Review of the Second Series of a Postgraduate Educational Television Series for Practicing Physicians in the State of Maine

GEORGE J. ROBERTSON, M.D.,* HAROLD F. PYKE, JR.** and EDWARD M. FRIEDLANDER†

Last year in *The Journal of the Maine Medical Association*[1] the authors reviewed the BOSTON MEDICAL REPORTS project following its first year of operation as an effort in postgraduate education for physicians via open circuit television. They reported that a study of efforts to develop a viewing audience in the State of Maine indicated that an audience of sufficient size and interest had been achieved to justify the effort and expense involved. Evaluation of audience interest in the initial series showed that a preponderance of Maine physicians who viewed any part of the series were enthusiastic and described the programs they saw as interesting, informative and professionally useful.

For the second year of operation of the series, the Bingham Associates Fund received support from the U.S. Public Health Service in the form of a three-year Community Health Services Grant. The second series of 24 programs, twice the number of the first year, was begun on November 3, 1964 over the four stations of the Maine Educational Television Network. Announcements of the first 12 programs of the new series, which followed the format established the previous year, were produced and mailed in advance of the first program to all Maine physicians. These were followed with monthly mailings which, in addition to a specially printed calendar indicating the dates and times each program would be shown, included a listing of references and an abstract of the content of each program. In addition, pages on which notes could be made were included. All material was punched to fit the 3-ring "Physician's Workbook" previously supplied to each physician as a container for all materials and, as a result, a reference for their later use.

The series began one week later for Massachusetts and New Hampshire physicians over the two educational television channels in those states. These programs were also aired twice weekly. Announcements were produced and mailed by the Postgraduate Medical Institute to physicians in those two states and Rhode Island.

Of the total of 24 programs scheduled for the new series, 11 were new productions, 8 were repeats of programs from the initial series, and 5 were selected from those produced by the University of Utah College of Medicine. All were chosen by the Curriculum Committee of the Postgraduate Medical Institute in collaboration with the Television Committee of the Maine Medical Association.

The 11 new programs were produced at the Tufts-New England Medical Center and at the studios of WGBH-TV in Boston. The subject material was chosen with the idea of developing "core curricula" which was started during the first year of the series. In this way, the basis for future specific course material for use as postgraduate educational programs in community hospitals could be developed. For example, the initial series featured a program entitled "Diagnosis and Management of Rheumatoid Arthritis." The second series added a program on "Gout: Diagnosis, Pathogenesis and Treatment." Planned for a future series is a program on rheumatic fever and collagen diseases.

As an interim step toward making all programs available, a "Catalog of Available Films and Videotapes" was developed at the end of the second series. These

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**Television Project Coordinator, Bingham Associates Fund.
†Director of Public Relations, Tufts-New England Medical Center and Consultant in Public Relations to Bingham Associates Fund.

catalogs were printed and mailed to directors of medical education, chiefs of staff, administrators and nursing directors in all Maine hospitals.

All programs of the second series were telecast throughout New England on Tuesdays at noon and repeated on Thursdays at 10:05 p.m. in Maine, and 11 p.m. elsewhere in New England. This change from the preceding year was made after a review of the initial evaluation which showed that physicians would view the programs during regularly scheduled noon conferences. In 14 community hospitals in Maine, groups of physicians would meet for lunch, watch the programs, and discuss the material immediately following the broadcast. These included: Augusta State, Augusta General, Bath Memorial, Cary Memorial, Fairview, Franklin County Memorial, Knox County, Madigan Memorial, Maine Medical Center, Mercy, Pineland, Rumford and Thayer Hospitals, and the Veterans Administration Hospital in Togus. The noon programs were limited to 30 minutes but the evening programs included an additional 10-minute segment of questions and answers with the participants.

In Massachusetts, another means of encouraging local participation was developed by Dr. Norman S. Stearns, Medical Director of Postgraduate Medical Institute. Immediately following the noon telecast, physicians in selected community hospitals were invited to participate in a two-way telephone conference, simultaneously carried on FM radio. Using this medium, the listening audience could ask questions of the television faculty and discuss the case material with them. Although only a limited number of locations could be included in this network at any one time, physicians in other hospitals could listen on FM radio and telephone questions into the panelists for further discussion.

An evaluation study of the second series has been completed. Its objectives were to determine if the viewing audience was being maintained and to demonstrate knowledge acquired from the programs. Responsibility for the study was again given to Dr. Ezra Saul and Mr. Richard Carter of the Institute for Psychological Research of Tufts University.

Initial studies done at the Annual Meeting of the Maine Medical Association to assess audience participation were disappointing. Physicians attending were requested to indicate on a printed listing the programs they remembered seeing, and whether they had seen them at their hospital or at home. The response rate of only 4% of registrants was dismally disappointing as compared to an 80% response rate the preceding year. This difference was attributed to the fact that a long and more complicated questionnaire requiring an inordinate amount of time to complete was used in place of the simple card form used previously.

Similarly, personal interviews were conducted in an effort to get additional information. Only 20 physicians attending the meeting completed the interview.

A repeat effort conducted by mail following the Annual Meeting resulted in a much better return. The same long form used at the meeting to assess the number of programs seen was mailed to all Maine physicians, and an interview form included to every third physician. Approximately 35% of the viewing questionnaires were completed and returned, and nearly 21% of those receiving interview forms completed and returned them with their completed viewing questionnaire. This return produced a valid sampling of the M.D. audience for the second series.

To attempt to evaluate the osteopathic physicians watching the series, a short form questionnaire was distributed to those registering at the Annual Meeting of the Maine Osteopathic Association. Nearly 100% return was realized from this group, providing a valid measure of this particular segment of the series' audience.

The plan developed to determine the extent of acquired knowledge was to request volunteers from among the physician audience who would submit to a series of tests involving three specially selected programs. Questions, designed to test for prior knowledge of the subjects being presented, were mailed to one group of volunteers prior to each telecast. Question material requiring recall of specific items of information presented in each of the three programs was mailed to all volunteer physicians following the telecasts.

Unfortunately, the evaluation consultants deemed the results of these tests unreliable because of the small sample size, the inadequacy of the test instrument, and the problems in matching the pre- and post-test participants satisfactorily. No further work was done in this study area.

Although some of the samples were small, the evaluation team was able to draw the following general conclusions from the combined studies:

1. BOSTON MEDICAL REPORTS has a substantial audience among physicians in the State of Maine. Between 77 and 90% of respondents indicated that they had seen at least one BOSTON MEDICAL REPORTS program of the second series.
2. The average physician watched from 7.2 to 10 of the 24 programs in the series. This compares favorably with the 3.34 of 12 programs reportedly seen in the first series.
3. An average of 84% of the programs were seen at home, the remainder were seen in hospitals or offices.
4. Nearly two-thirds of the respondents preferred Thursday night broadcasts to those on Tuesday noon.
5. Approximately 25% of physicians interviewed, and those returning census cards, reported incidents of learning.

In terms of fulfilling its objectives, the evaluation study of the 1964-1965 series produced mixed reactions. There is little doubt that the objective of maintaining an audience had been accomplished. In fact, it would appear that a greater number of physicians were watch-

ing more programs in the second series than in the first. The problem of determining the extent of acquired knowledge or of attempting to ascertain specific incidents of learning from the television programs, however, was a much more difficult one to solve. Since improvement of medical care is the ultimate objective of this total project, primary evaluation efforts during the next two years will focus on methods of assessing changes in physician behavior as a result of information gained from the BOSTON MEDICAL REPORTS series.

Two more years of BOSTON MEDICAL REPORTS remain under the Public Health Service grant. Following its expiration, some mechanism has to be found to continue to provide financial support for the project. The problem of continuing support is also being faced by similar postgraduate efforts and organizations in other sections of the country. With this in mind, 12 producers of television programs for continuing medical education met at the University of South Carolina in March of 1965 to discuss the formation of a national program service. In the fall of 1965, the Association of Medical Television Broadcasters was officially organized.

The objectives of this organization are to promote the use of broadcast television as a medium for continued education through collaboratively-produced programs, to make available and distribute videotapes on a national basis, and to attempt to improve production techniques and effective teaching techniques through periodic workshops.

Through the workings of this organization, the knowledge and resources of medical centers throughout the country may be made available to the practicing physician in his community and in his own hospital.

In its first two years, BOSTON MEDICAL REPORTS has demonstrated that open-circuit television can be an effective way to bring instruction to the practicing physician.

Continued cooperation with the Maine Medical Association, together with constructive criticism from its members, gives direction and meaning to these efforts. It is the hope of the authors that BOSTON MEDICAL REPORTS will continue to play an ever-increasingly important role in continuing medical education in the State of Maine.

REFERENCE

1. Robertson, G. J., Pyke, H. F., Jr., Friedlander, E. M.: BOSTON MEDICAL REPORTS. *J. of Maine Med. Assn.* 3: 41-43, March, 1965.

Population Expansion One of 6 Topics of General Sessions at 1966 AMA Annual Convention

Population expansion, a subject much discussed in both medical and lay circles, will be one of six topics presented in general scientific sessions at this year's Annual Convention of the American Medical Association.

The Convention is to be held in Chicago, June 26-30; the Scientific Program will be at McCormick Place, and the House of Delegates will convene at the Palmer House.

Scientific Sections of the AMA Council on Postgraduate Programs taking part in the General Meeting on population expansion will be Preventive Medicine, General Practice, Internal Medicine, Obstetrics and Gynecology, and Pediatrics. The General Scientific Meetings are open to all physicians attending the Annual Convention.

Other General Scientific Meetings on this year's Annual Convention program will be on the subjects of: emphysema, burns, mysterious fevers, community hospital coronary care units, and headache.

In addition to the General Sessions, each of the 23 Scientific Sections will present scientific programs. Many of the Section programs will, as in past years, be joint meetings of two or more Sections and, in some instances, a specialty society.

The entire Scientific Program for the 1966 Annual Convention will be published in the May 9 issue of the Journal of the American Medical Association.

Calculous Cholecystitis in an 11-Year-Old Girl — Report of a Case

GILBERT R. GRIMES, M.D. and JAMES V. O'SULLIVAN, M.D.

Gallbladder disease in children, once considered an uncommon disease, is now being diagnosed more frequently.

Brenner & Stewart[1] reviewed the literature and added a further 244 cases of childhood gallbladder disease to the 326 already reported.

A case of calculous cholecystitis is herein reported. The case is of interest in that this child first presented with a history and physical findings entirely consistent with gallbladder disease and extrahepatic biliary obstruction.

CASE REPORT

B. B. an obese 11-year-old (115 lbs., 48 inches) white female child was admitted to the Central Maine General Hospital on May 17, 1965, complaining of abdominal pain of three days' duration and associated jaundice. The pain was situated in the epigastrium and right upper quadrant with radiation to the right subscapular region and also to the left upper quadrant. It was described as continuous. There was associated nausea and vomiting. The urine was noted to be dark in color and the stools were light.

Intermittent abdominal pain had been noted over the year prior to admission, with gradually developing intolerance to fatty foods. The past history and system review were normal. There was a familial history of gallbladder disease in two maternal aunts. There was also a family history of exogenous obesity in siblings and parents.

Physical examination on admission revealed a temperature of 100°, pulse 110. Slight scleral icterus was present. There was marked abdominal tenderness, and spasm most marked in the upper abdominal quadrants, and with maximal tenderness in the right upper quadrant. There was no palpable mass. The liver and spleen were not enlarged.

Urinalysis showed 4 plus bile. Hemogram showed a WBC of 16,500 with 87 mature neutrophils, 5 stabs, 3 lymphs and 6 monos. Hemoglobin was 14.9 and hematocrit 43.5. Following hydration the hemogram reverted to normal.

Total bilirubin on admission was 4.0 mgs.%; Direct 3.1; Indirect 0.9 mgs.%. BUN 6.2 mgs.%. Alkaline phosphatase 16.2 Bodansky units. Prothrombin time 100%. Thymol turbidity 0.7 units. Cephalin flocculation normal. Serum transaminase 380 units.

The child was treated conservatively for five days until there was satisfactory clinical and chemical improvement.

Oral cholecystogram was performed five days after admission when the serum bilirubin had fallen to 1.0 mgs.% total. The Graham test showed no satisfactory visualization of the gallbladder and no stones were demonstrated.

At operation on May 26, 1965, under general anesthesia the abdomen was entered via a right upper quadrant paramedian incision. The gallbladder was found to be thick-walled and contained multiple stones averaging 6-8 mms. in diameter. On opening the gallbladder following its removal, multiple smaller seedlike calculi were noted. The common duct did not contain palpable stones but was somewhat thickened and measured approximately 1 cm. in diameter. In view of the findings of small stones and thickened common duct, along with the history of jaundice, exploration of the common duct was performed with negative findings. The sphincter of Oddi was lax, permitting easy passage of a #7 dilator. The pancreas was normal to palpation.

The cecum and appendix were in the right lower quadrant and routine appendectomy was performed. A short limb T-tube was placed in the common duct and removed on the twelfth postoperative day.

The postoperative course was uneventful. A T-tube cholangiogram performed prior to removal of the T-tube showed good filling of the hepatic radicles and common duct and normal emptying into the duodenum. There was no evidence of retained calculus.

The child has remained asymptomatic since surgery. The pathologist reported marked chronic cholecystitis with cholelithiasis. The appendix showed mild chronic appendicitis.

It is interesting that the incidence of jaundice in cholecystic disease is greater in children than in adults, but there is a lower incidence of common duct stone.

In children the duration of the disease is shorter, whilst the chronicity with recurrences and exacerbations, often over a period of many years, is characteristic of the adult form.

With regard to symptoms, abdominal pain is reportedly not localized in the childhood form of cholecystitis and referred pain to the shoulder or back is not as common as in adults. In the case reported above, however, the signs and symptoms were identical with those seen in the adult.

The six causative factors in childhood biliary tract disease are: 1. infection and infestation; 2. anatomic; 3. constitutional; 4. metabolic; 5. hereditary and 6. hemolytic anemias. The constitutional factor of obesity appears to be a common finding and a family history of gallbladder disease is frequently associated with childhood cholecystitis.

The differential diagnosis may frequently be difficult unless the symptoms directly suggest gallbladder disease as in this reported case.

Acute cholecystitis in children has been diagnosed as acute appendicitis, intussusception, hepatitis, perforated viscus with peritonitis, and hepatoma. It is easy to see that recurrent mild attacks of cholecystitis may be easily missed unless cholecystogram is performed more frequently in such cases.

NOTE: A 10-year-old sister of the above patient, also markedly obese, had a negative cholecystographic study.

CONCLUSION

A case of calculous cholecystitis with extrahepatic biliary obstruction has been described in an 11-year-old girl. The treatment consisted of cholecystectomy with common duct exploration. The importance of cholecystographic examination in suspected cases is emphasized.

REFERENCE

1. Brenner, Richard W., and Stewart, Charles F., "Cholecystitis in Children," *Review of Surgery*, Vol. 21, December, 1965.

Dr. Grimes, 185 Webster Street, Lewiston, Maine
Dr. O'Sullivan, Central Maine Gen. Hosp., Lewiston, Maine

Who is Going to Care for the Sick?

PAUL H. PFEIFFER, M.D.

The title is purposely ambiguous. It is meant to imply that doctors will soon be so preoccupied with other matters that they will be unable to care for the sick and at the same time may become so involved in a multitude of responsibilities that they will need help themselves.

At a recent breakfast meeting in Philadelphia held at the time of the Clinical Convention of the American Medical Association, several of us heard a discussion of "The Prevention of Long-Term Illness" which led me to compose this article. Two erudite clinicians, at this session, presented the idea that illness could best be prevented by a careful health appraisal in the doctor's office–to be repeated annually. An hour to an hour and a half would be required to perform the health appraisal generating so much patient satisfaction that if the patient were not permitted to return a year later (to the day) for his reappraisal, he would feel neglected. If this plan develops, and it has much to recommend it from the point of view of the physician as well as the patient, doctors' offices would be booked up from one year to the next with the six to eight to ten patients he would see per day. Assuming that the doctor still has a little "free" time after he has taken care of the health appraisals (with lectures to the obese on the blessings of dietary continence, advice to the coughers on the evils of tobacco, the neurasthenics on muscular fitness, the indolent "boob-tube" gazers on how to find intellectual stimulation and so on) what is he doing with this time?

The physician must in due course meet his hospital obligations by serving on the several committees required by the Committee on Accreditation. There are tissue committees, record committees, audit committees, patient-care committees, pharmacy committees, admission and discharge committees, research, library, infections and dietary committees and others as well as the all-important Utilization Review Committee. He also has obligations to his county and state medical society and the various committees organized for their effective function. He may even be one of the select few to be appointed a delegate to the American Medical Association and be required to travel to several parts of the country in order to attend national conventions and perhaps also serve on committees meeting during the interim. Not only the specialists but the generalists have their own organizations with attendant meetings. There are organizations dedicated to the eradication of tuberculosis, heart disease, cancer and a multitude of other diseases and conditions and these have committees usually blessed with physician members.

As Osler correctly pointed out a dedicated physician must continue his medical education throughout his productive life. In order to accomplish this there are staff meetings at every respectable hospital occurring at least once a month and possibly as often as once a week, supplemented with county, state and national society educational meetings not to mention the specialty and general practice sessions. The hospital is the site of clinical, radiological and pathological conferences which can be supplemented with journal clubs and television and radio hookups with distant medical centers. Libraries and a multitude of medical journals are at the beck and call of the physician who feels he is falling behind in the ever-mounting deluge of new knowledge in his profession.

Aside from the above pressing inroads on the MD's leisure there are community obligations such as commissions to study local hospital and nursing needs and the recently described Debakey Commission's report to set up regional centers for training in Heart disease, Stroke and Cancer. There are speakers, preferably MD's, needed to address local service clubs, veterans' associations, and those for clergymen, undertakers and dentists on matters of public concern in the field of medicine. There are also health lectures to be given or supervised at the school and college level. The Alcoholics Anonymous, Dial-a-Psychiatrist or a Dietitian all need physician support.

The advent of "third party" medicine has caused the physician to become more and more involved in economic matters requiring committee work with insurance companies, state departments of health and welfare and the National Social Security Administration. And in line with this activity the physician is urged to get nvolved in local politics to see that those friendly to organized medicine are elected.

The community expects the physician to lead an exemplary family life. A good churchman, ideal father and husband he tries to spend time at those family activities which will cement the bonds of a happy family relationship.

In conclusion, one gains the impression, rightly or wrongly, that certain and varied demands are being made on the physicians' leisure time in the mad, mad world we live in. Serious consideration is now being directed to have MD's spend a good part of their working day doing health appraisals of asymptomatic people. It may seem like a rhetorical question but who, pray tell, will tend the sick? Don't look now, but IBM is making some pretty smart machines.

14 Gilman Street, Waterville, Maine

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Malaria, 1966

CHARLES H. OKEY, Ph.D.*

An increase in the number of malaria cases diagnosed in the United States is the cause for growing concern among health authorities, both military and civilian. Concern of military authorities stems from the high percentage of cases diagnosed among personnel recently returned from Southeast Asia. Almost 60% of the 130 cases in 1965 were military cases and about one-third were discovered during the last quarter of the year when increasing numbers of servicemen were returning to the United States from duty in Viet Nam. Civilian health authorities are following the situation closely and are alerting physicians to watch for cases among their patients, particularly persons returning from overseas. Maine physicians are asked to cooperate with the Department by reporting cases. Facilities of the Public Health Laboratory (Diagnostic Laboratory) for malaria diagnosis by examination of peripheral blood smears are available.

A Malaria Surveillance unit has been created at the Communicable Disease Center in Atlanta to collect epidemiologic data, disseminate therapeutic information and provide diagnostic assistance. Information regarding therapy is of particular importance in that increasing numbers of cases of falciparum malaria in military personnel in Southeast Asia have exhibited drug resistance. Material provided by the unit is quoted in the following paragraphs.

"Considerations of Diagnosis and Therapy of Plasmodium *Falciparum Malaria*

The Department of the Army has submitted an editorial to *The Journal of the American Medical Association* concerning the need for physicians to be alert to the possibility of falciparum malaria developing in individuals who have traveled through or served in Southeast Asia. They warn that 'presenting symptoms may include gastrointestinal or musculoskeletal complaints, unexplained high fever, postural hypotension, medical shock, irrational behavior or delirium. Rapid diagnosis and initiation of therapy are of the utmost importance. Because routine thin blood smears are less apt to show the parasite in falciparum than in other forms of malaria, the use of repeated thick smears is advised in any patient with an obscure illness with a background which raises the potentiality of malaria infection.'"

"Chemotherapy–Falciparum Malaria

The following is an outline for current treatment of falciparum malaria adapted from a guideline prepared by the Department of the Army, Office of The Surgeon General, and modified and supplemented by recommendations by the Laboratory of Parasite Chemotherapy, National Institute of Allergy and Infectious Disease, National Institutes of Health, Bethesda, Maryland. This outline is presented here because of the increasing possibility that resistant malaria will be encountered in domestic medical practice as a result of the increasing numbers of servicemen returning from South Viet Nam. It should be noted that resistance of *P. falciparum* to chloroquine has been confirmed only in certain areas of Colombia, Brazil, Cambodia, Malaya, South Viet Nam and Thailand.

"1. Probably in most cases of previously untreated falciparum malaria, the standard chloroquine regimen as outlined in the booklet *Control of Communicable Disease in Man,* 10th Edition, 1965, an official report of the American Public Health Association, will prove effective. This consists of an initial dose of chloroquine phosphate, 1.0 gram (600 mg. of base), followed by 0.5 gram (300 mg. of base) six hours later, and then 0.5 gram once daily for the next two successive days for a total dose of 2.5 grams (1.5 grams of base) of chloroquine phosphate in three days. If the patient is unable to take or retain chloroquine given orally, the drug may be given as the hydrochloride, 250 mgs intramuscularly, and repeated in six hours, switching as soon as possible to the oral medication. This regimen should be employed initially for patients with any type of malaria who are not critically ill and for whom the history does not indicate that such a course of therapy has already been administered.

"2. In patients with *P. falciparum* malaria who (a)

*Director, Public Health Laboratory, Augusta, Maine.

are gravely ill or (b) have not responded to the chloroquine treatment described above or (c) have a recrudescence despite a previous response to a course of chloroquine, the treatment of choice is quinine. This is administered as quinine sulfate 0.65 gram (two tablets of 5 grains each) every eight hours for seven to ten days. Because of a variable tendency to develop postural hypotension, patients should be kept at bed rest during quinine administration. The urine output should be measured and if oliguria develops, quinine should be temporarily discontinued. During oliguria, quinine blood levels may rise precipitously and acute quinine toxicity may ensue.

"3. If the patient is unable to retain quinine because of vomiting, or when coma, presumed due to falciparum malaria, is present, the drug may be given intravenously. It must be emphasized that this treatment carries a *serious hazard* and should be resorted to only when the patient's condition clearly warrants the risk and no other form of treatment is possible. Quinine is administered intravenously as the dihydrochloride, 600 mg in 600 cc of normal saline, by *very slow* intravenous drip with constant monitoring of the blood pressure and the pulse to detect hypotension or arrhythmia. The same intravenous dose may be repeated at intervals of eight hours if the patient's condition requires. Oral therapy, by stomach tube if necessary, should be utilized as soon as possible.

"4. Patients who respond to quinine therapy but later undergo recrudescence should be retreated with quinine as described above. If necessary, the course of quinine may be maintained for 21 days at a dosage of 0.65 grams (10 grains) every eight hours, as tolerated.

"5. If asexual parasites persist during quinine therapy, regardless of the clinical response, the patient should receive a course of pyrimethamine (Daraprim®), 50 mg. daily for *three days* concurrently with sulfadiazine, 0.5 gram every six hours for five days. Careful attention should be given to maintaining adequate hydration during this therapy."

Four cases are described in the material issued by the Malaria Surveillance unit to demonstrate the clinical and epidemiologic features of malaria as seen today.

Case No. 1

R.J.C., a 24-year old Caucasian male from Philadelphia, Pennsylvania, was inducted into the Army in December, 1963, and transferred to Viet Nam in August, 1965. He departed his combat area in preparation for discharge on the 4th of December, 1965. On December 7th, en route to Travis Air Force Base in California, he became nauseated, vomited twice, and had a slight chill. On December 9th, in Oakland, California, he was seen on sick call with symptoms consistent with an upper respiratory illness. On December 12th he was discharged from the service. He returned to Philadelphia and remained at home for 2 days. However, he became increasingly ill and was referred to the Temple University Hospital with a temperature of 105° on December 14th. The diagnosis of falciparum malaria was made, and 138,000 parasites per cubic mm. were noted. The patient received quinine sulfate, 960 mg. orally daily for 2 days and on the third day Paludrine (proguanil), 0.3 gm. twice daily was started and continued through the remainder of his hospitalization. His clinical condition was satisfactory throughout his hospital course. However, he remained febrile and his parasite count increased, reaching 588,000 per cubic mm. on the 7th hospital day. Nevertheless, he remained alert until the 8th hospital day when he suffered a cardiac arrest and expired. The patient allegedly took chloroquine-primaquine prophylaxis regularly, and at the time of discharge was given this medication to take for 6 weeks.

Case No. 2

J. T. is a 24-year-old Caucasian male who left the combat area on November 29th to return to the United States. On December 5th, at the U.S. Army Personnel Center in Oakland, California, he experienced chills, fever, and diarrhea. On December 6th he was seen at the dispensary where blood smears were negative for malaria. Following discharge on December 9th, he went to Fort Collins, Colorado, where, on December 10th, he experienced chills, fever and diarrhea again and developed vomiting. After a 3-day hospitalization without diagnosis, he was transferred to the Veterans Administration Hospital in Denver on December 13th. Blood smears showed *P. falciparum,* and the patient responded to therapy with quinine, 600 mg. orally every 8 hours for 14 days, and chloroquine, 500 mg. (300 mg. base) daily for 14 days. He stated he had taken malaria prophylaxis regularly.

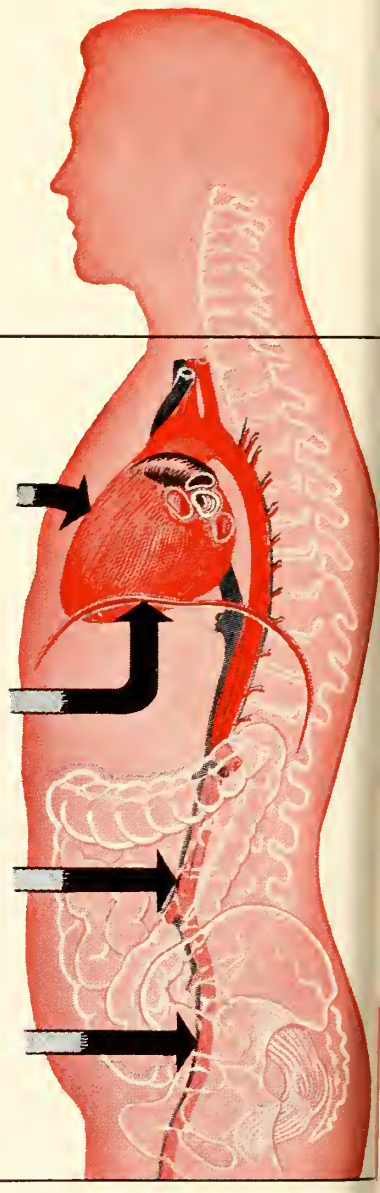
Case No. 3

N. L., a 24-year-old Caucasian male, departed the area of combat on December 3rd. He arrived in Oakland, California on December 6th and remained there through his discharge on the 9th. He returned home the following day and developed symptoms which he attributed to the "flu". On December 11th he developed severe chills, fever, and headache and was hospitalized in Burlington, Colorado. Falciparum malaria was diagnosed, and he was transferred to Fitzsimons Army Hospital in Denver. There he was treated with quinine 1 gram every 8 hours for 2 days and 0.5 grams every 6 hours for 28 days. Despite a complicated clinical course including the development of severe intravascular hemolysis and acute renal failure, he is now doing well. Peripheral blood smears revealed 50-75% infection rates of red blood cells. He allegedly took his chloroquine-primaquine phophylaxis regularly.

Case No. 4

R.E.N. was a 24-year-old Caucasian male who was discharged in Oakland, California on December 5, 1965, and arrived home the following day in Mancos, Colorado. At that time he was experiencing chills and fever. On December 9th his fever rose to 105° and he developed severe chills, generalized muscular aching and diarrhea, and was hospitalized in Cortez, Colorado. Despite antipyretic therapy, he continued to experience daily fever spikes to 105° and, on December 13th, he received quinine 2 grams orally followed by 1 gram every 8 hours. The following day 80% of his red blood cells were noted to be parasitized with *P. falciparum.* On December 15th, he developed intravascular hemolysis, and became oliguric with a rising NPN. Chloroquine, 500 milligrams daily, was started. Inclement weather prevented a transfer to Fitzsimons Army Hospital in Denver. Therapy included blood transfusions, heparin administration, mannitol diuresis, and peritoneal dialysis. His condition continued to deteriorate, and he expired on December 17. The history of chemoprophylaxis was unavailable in this case.

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SEARLE

Research in the Service of Medicine



AMERICAN CANCER SOCIETY - MAINE DIVISION. INC.

Brunswick, Maine

Blood Test to Determine Response to Drugs

A blood test which, in preliminary trials, has shown whether a cancer patient will respond to drugs has been developed by scientists at the Albert Einstein Medical Center and the Moss Rehabilitation Hospital.

The American Cancer Society recently reported the results achieved by Drs. Seymour Winsten, Stanley Levick and Edwin Cohn after several years of experiments.

The group uses an apparatus–a large scale micro-glass bead continuous flow electrophoresis–which enables them to separate simultaneously a half-dozen or more enzymes in a single blood serum sample. Enzymes are catalysts which spark innumerable chemical reactions in the cell and in the body.

One enzyme in particular showed interesting shifts. In some patients, the enzyme's activity resided largely in a blood protein called gamma globulin, the stuff from which antibodies are made; in other patients measurable activity could be found in different blood proteins such as albumin and alpha globulin. The enzyme is PHI, or phosphohexoseisomerase.

Close checking showed that there was a distinct difference between patients with the PHI activity in the blood fractions.

Patients with measurable activity of PHI in the albumin and alpha globulin proteins usually responded to drug treatment.

Patients with PHI activity almost exclusively in the gamma globulin fraction, on the other hand, never did show a really good response to anti-cancer drugs; any good reaction was extremely brief.

As drug resistance developed in the once-responsive cases, the blood pattern changed accordingly.

Patients with non-cancerous diseases of the liver showed PHI activity in both fractions, but with considerable activity in the albumin-alpha globulin area.

Tests have been conducted so far on 190 cancer patients and 57 non-cancerous people.

The scientists are now trying to obtain PHI in pure form so they can determine what the enzyme does in health and in disease and why there is a shift from one serum protein to another as drug-resistance develops in cancer.

From American Cancer Society News Service Release

Reserve these dates



for

the 1966 Annual Session

Maine Medical Association

The Samoset, Rockland, Maine

The Scientific Program includes . . .

- "Management of Biliary Tract Disease and Associated Conditions" — A panel including the following participants
  Kenneth W. Warren, M.D., Lahey Clinic Foundation
  Robert E. Wise, M.D., Lahey Clinic Foundation
  Perry J. Culver, M.D., Massachusetts General Hospital

- "Medical Emergencies" — A panel to be presented by Bangor area physicians
  Richard C. Wadsworth, M.D., Producer-Director
  George W. Wood, III, M.D., Moderator
  Lloyd Brown, M.D.
  Warren G. Strout, M.D.
  Edward B. Babcock, M.D.
  Harold D. Cross, M.D.

- "The New Concepts of Diabetes"
  Leo P. Krall, M.D. of Boston

- "Diseases of the Esophagus" (Sponsored by Veterans Administration Center, Togus)
  Julius Wolf, M.D. of New York

Programs to be sponsored by —
Maine Medico-Legal Society
Maine Trauma Committee
Maine Chapter, American College of Surgeons
(Speakers to be announced)

Specialty Group Meetings:

Maine Society of Clinical Hypnosis
Maine Thoracic Society
Maine Society of Internal Medicine
Section on Ophthalmology of the Maine Medical Association
Maine Society of Anesthesiology
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Maine Society of Obstetrics and Gynecology

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Specialty Group Programs
Program For The Ladies
Reservation Forms

Maine Heart Association Notes
116 State Street, Augusta, Maine

The Rebound Phenomenon — Fact Or Fancy?

Experience with Discontinuation of Long-Term Anticoagulation Therapy after Myocardial Infarction

"There is considerable controversy regarding the recurrence of thromboembolic episodes following discontinuation of long-term anticoagulant therapy. . . . The question of a rebound state of hypercoagulability, especially after gastrointestinal bleeding necessitating stopping of anticoagulants, has been commented upon both in clinical and experimental studies. . . .

This paper reports the results of a prospective study in which long-term anticoagulant therapy for myocardial infarction was either abruptly stopped or gradually tapered. It is aimed at examining the existence of a clinical state of rebound hypercoagulability.

Long-term anticoagulant therapy was gradually tapered over a six-week period in 63 patients, and stopped abruptly in 71. . . . A careful follow-up showed that there was no difference in the incidence of thrombotic events in the first six weeks after discontinuation of therapy, whether this was stopped abruptly or gradually These results suggest that clinically recognized "rebound" thrombosis does not occur after long-term coumadin therapy is stopped. There appears to be no hazard inherent in stopping long-term coumadin therapy abruptly. A possible exception to this is patients whose therapy must be stopped because of bleeding episodes."

Submitted by Jacob B. Dana, M.D.
Reference: Van Cleve, R., M.D., Circulation, Volume 32, pages 878-880, 1965

Necrologies

Philip P. Thompson, Sr., M.D.
1881-1966

Philip P. Thompson, Sr., M.D., 84, of South Portland, Maine, retired physician and surgeon and veteran of World War I, died on February 16, 1966. He was born in Portland, Maine on April 29, 1881, son of Frederick W. and Lillie Dean Pickering Thompson.

Dr. Thompson graduated from Dartmouth College in 1902, attended Bowdoin Medical School one year, received his medical degree from Johns Hopkins University School of Medicine in 1906 and interned at Carney Hospital in Boston.

Dr. Thompson was a former member and past president of the Cumberland County Medical Society, a former member of the Maine Medical Association, the New England Surgical Society, the Portland Medical Club and a fellow of the American College of Surgeons. He served many years on the surgical staff of the Maine General Hospital, was chief surgeon at the United States Marine General Hospital in Portland, a consulting surgical staff member of Webber Hospital in Biddeford, served on the original staff of Queens and Mercy Hospitals in Portland and was a long-time member of the staff of the Maine Eye and Ear Infirmary.

Surviving are his widow, the former Jennie Means; two sons, Dr. Philip P. Thompson, Jr. of Portland and John F. Thompson of South Portland; a daughter, Mrs. Philip Tukey of Bangor and eleven grandchildren.

Stanwood E. Fisher, M.D.
1878-1966

Stanwood E. Fisher, M.D., 87, of Portland, Maine, retired physician and surgeon, died on February 23, 1966. He was born in Portland, Maine on September 2, 1878, son of Thomas and Annie Constable Fisher.

Dr. Fisher attended local schools and received his medical degree from Bowdoin Medical School in 1906. He did postgraduate work at the Maine General Hospital, the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital.

He was a former member of the Maine Medical Association, the Cumberland County Medical Society, the American College of Surgeons and was an associate surgeon at the Maine Medical Center for several years.

Surviving are his widow, the former Frances Darker; a son, Stanwood E. Fisher, Jr. of Fort Lauderdale, Florida; two daughters, Mrs. Frank J. Mitchell of Sherborn, Massachusetts and Mrs. Richard Y. Chadwick of Cumberland Foreside and eight grandchildren.

County Society Notes

HANCOCK

A meeting of the Hancock County Medical Society was held in Ellsworth, Maine on February 9, 1966.

A panel of four physicians discussed the current management of pulmonary diseases. William B. Grow, M.D. of the Central Maine Sanatorium demonstrated the infectious communicability of tuberculosis. John F. Reynolds, M.D. of Waterville spoke on the surgical management of pulmonary tuberculosis. Richard T. Chamberlin, M.D. discussed the classifications and treatment of chronic obstructive emphysema and H. Richard Hornberger, M.D. spoke on the recognition and results of treatment of pulmonary malignancies.

Russell G. Williamson, M.D., *Secretary*

FRANKLIN

The following members of the Franklin County Medical Society have been elected to serve as delegate and alternate to the Maine Medical Association House of Delegates:

Delegate: Hays G. Bowne, M.D., Farmington. Alternate: Paul A. Brinkman, M.D., Farmington

Maynard B. Colley, M.D., *Secretary*

SOMERSET

Members of the Somerset County Medical Society who have been elected to serve as delegates and alternates to the Maine Medical Association House of Delegates are as follows:

Delegates: Drs. Richard P. Laney, Skowhegan and H. Carl Amrein, Madison. Alternates: Drs. Harland G. Turner, Norridgewock and Edgar J. Smith, Fairfield

LINCOLN-SAGADAHOC

The Lincoln-Sagadahoc County Medical Society met at The Ledges in Wiscasset, Maine on February 15, 1966. The meeting was called to order by the President, Alan W. Zeller, M.D.

Francis A. Winchenbach, M.D. stated that the fiscal intermediary for Plan A of the Medicare Act will be the Associated Hospital Service of Maine, and the fiscal agent for Plan B will be the Union Mutual Life Insurance Company in Portland. All members were urged to speak to patients recommending that they avail themselves of both plans.

Mr. Barry Smith director of Speech and Hearing at the Hyde Rehabilitation Center in Bath and his associate Miss Nelson, were the guest speakers. They gave an excellent presentation of speech and hearing problems and their handling.

George W. Bostwick, M.D., *Secretary*

KENNEBEC

Twenty-four members were present at the meeting of the Kennebec County Medical Association held at the Fenway Motor Motel in Waterville, Maine on February 17, 1966. The President, Allan J. Stinchfield, M.D. called the meeting to order.

Richard H. Dennis, M.D. requested that members interested in serving on committees of the Maine Medical Association get in touch with M.M.A. headquarters at P.O. Box 637 in Brunswick.

Mr. John D. Dow, supervisor of the Services to Armed Forces Rejectees, spoke to the association and asked for our cooperation and comments in evaluating selective service system rejectees.

Dr. Dennis noted that the Union Mutual Life Insurance Company has been selected for Plan B of the Medicare program. Brinton T. Darlington, M.D. proposed that the association endorse a program proposed by the Waterville hospitals encouraging members of the community to join the physicians portion of Medicare which was seconded and passed by the association.

Theodore A. Potter, M.D., chief of Orthopedics at the Robert A. Brigham Hospital in Boston, spoke on the subject of surgery of the knee joint and chronic arthritis.

LUCIEN F. VEILLEUX, M.D., *Secretary*

CUMBERLAND

Eighty-one members and guests attended the meeting of the Cumberland County Medical Society which was held at the Holiday Inn in Portland, Maine on February 17, 1966.

A panel discussion on "The Dying Patient" was presented by the Reverend Panton, Drs. Stanley E. Herrick, Jr., Richard A. Levy, George F. Sager and Robert A. Bearor.

A letter was read from the Woman's Auxiliary to the society thanking the membership for the combined meeting in January. A letter was read from Mr. John D. Dow, supervisor of the Services to Armed Forces Rejectees, requesting guidance as to whom to refer rejectees who have no regular physician. It was voted the members of this society continue to cooperate in seeing such rejectees, utilizing the usual channels of communication for obtaining a physician's services and a letter to this effect will be sent to Mr. Dow.

Dr. Morrison announced receipt of resolutions recently adopted by the AMA and that they will be discussed with the membership at the next meeting. He announced a retreat in the near future sponsored by the Congregational Churches.

Dr. Morrison read a letter from Mrs. Kennard announcing the April 3 meeting of the House of Delegates of the Maine Medical Association, requesting any business for the meeting and also the names of persons who might be interested in serving on the various committees.

STANLEY B. SYLVESTER, M.D., *Secretary*

YORK

A meeting of the York County Medical Society was held at the Webber Hospital in Biddeford, Maine on March 9, 1966. The President, Walter R. Peterlein, Jr., M.D. called the meeting to order following a social hour and dinner.

Mr. Donald V. Little, Regional Representative of Kidder Peabody & Company in Boston, gave a very interesting talk on the current outlook of investments for capital gains.

The next meeting will be held on June 8, 1966 at the Notre Dame Hospital in Biddeford.

CHARLES W. KINGHORN, M.D., *Secretary*

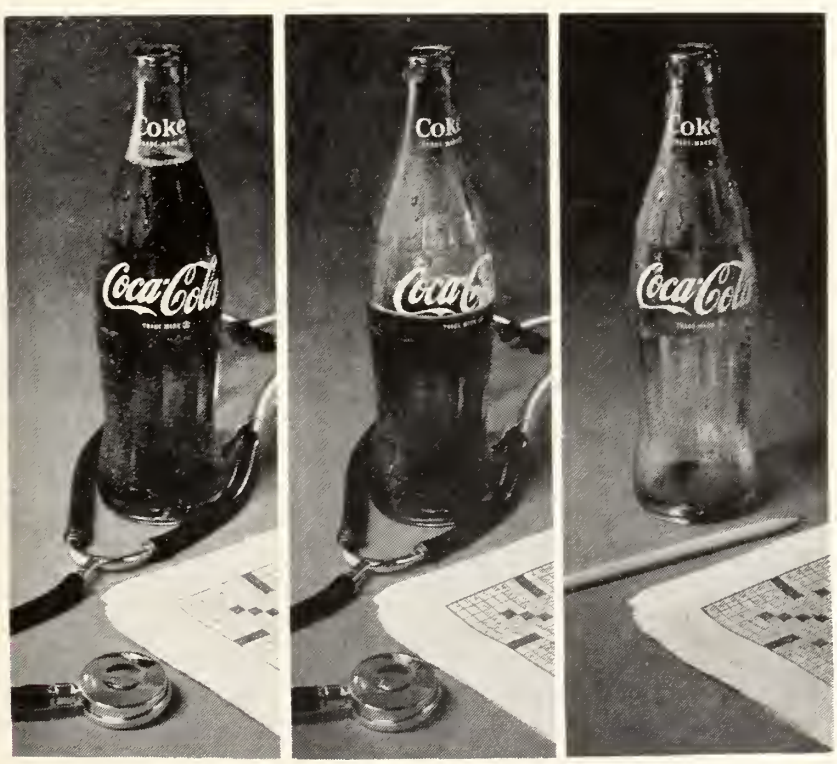
New Members

CUMBERLAND

Thomas F. Conneen, M.D., 131 Chadwick St., Portland
Behzad Fakhery, M.D., 66 So. High St., Bridgton

YORK

Woon B. Rhee, M.D., Branch B, Poughkeepsie, New York
Hyung J. Yeon, M.D., Branch B, Poughkeepsie, New York



11:47 pm 11:53 pm 12:06 am

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News, Notes and Announcements

Scientific Exhibits for the Annual Session of the Maine Medical Association Samoset Hotel, Rockland, Maine June 12-14, 1966

"With the aim of supporting and encouraging the development of scientific exhibits of high quality, Mead Johnson Laboratories has created the Aesculapius Award, designed to recognize and reward excellence in concept, originality and execution of scientific exhibit presentations at the Annual Session of the M.M.A.

"Under the terms of this program, the most outstanding scientific exhibit is selected by a committee of the medical society. The author of the winning exhibit is presented with an appropriate certificate and a cash prize of $200."

Applications for exhibit space must be sent no later than May 15, 1966 to the MAINE MEDICAL ASSOCIATION, P.O. BOX 637, BRUNSWICK, MAINE 04011.

Application may be made by informal letter, stating the nature and description of the exhibit and the amount of space required for it.

Complete information relative to setup of exhibits, etc., will be sent on receipt of application.

Rocky Mountain Cancer Conference

The Mile High City, Denver, Colorado, is once again the site of the 20th Annual Rocky Mountain Cancer Conference, July 15-16, 1966, at the Brown Palace Hotel. The two-day Conference will feature some of the nation's most distinguished speakers on the subject of cancer.

Dr. Charles L. Hudson, now President-elect of the American Medical Association and Dr. Leonard W. Larsen, President of the American Cancer Society, will participate in the Conference which is co-sponsored by the Colorado Division of the American Cancer Society and the Colorado Medical Society.

Speakers on the two-day scientific program include: J. Max Chamberlain, M.D., a Thoracic Surgeon from New York; Emerson Day, M.D., Director of the Strang Prevention Clinic in New York; Robert C. Horn, Jr., M.D., Director of Pathology, Henry Ford Hospital, Detroit; and Leo G. Rigler, M.D., Department of Radiology, Center for Health Sciences, Los Angeles.

Winner of Third Annual Maine "Ability Counts" Contest Announced

Cheryl L. Abbott, 17-year-old Waterville High senior, is the winner of first prize in the third annual Maine "Ability Counts" Contest sponsored for high school juniors and seniors by the Governor's Committee on Employment of the Handicapped.

Miss Abbott's community survey report on "What Handicapped Workers Are Contributing to My Community" was judged best from more than 100 entries. She will receive a $100 U.S. Savings Bond and an all-expense paid trip to Washington, D.C., when winners from all participating states will be guests at the annual meeting of the President's Committee on Employment of the Handicapped.

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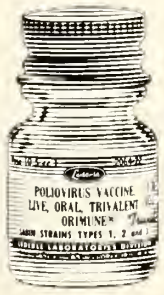
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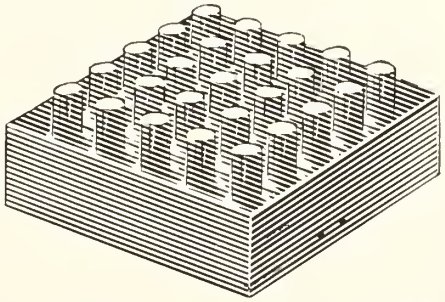


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Effectiveness—may be expected to confer active immunity against all three types of poliovirus infection in at least ninety percent of susceptibles only if given at full dosage, as directed. No characteristic side effects have been reported. There are, however, certain contraindications. These are, broadly: acute illness, conditions which may adversely affect immune response, and advanced debilitated states. In these, vaccination should be postponed until after recovery.
In infants vaccination should not be commenced before the sixth week of life. Do not give to patients with viral disease, or if there is persistent diarrhea or vomiting. ORIMUNE and live virus measles vaccine should be given separately.
Dosage—initial immunization: two doses each given orally at least 8 weeks apart. (Give a third dose to infants at 10-12 months). Booster immunization: one dose, given orally. See package literature for full directions.



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Side effects are possible but rare: vesiculation, ulceration or necrosis at test site. Contraindications, none; but use with caution in active tuberculosis. Available in boxes of 5 (new individually-capped unit); cartons of 25.

*Rosenthal, S. R., Nikurs, L., Yordy, E., and Williams, W.: Scientific Exhibit Presented at the Annual Meeting of the National Tuberculosis Association, Chicago, Illinois, May 30-June 2, 1965.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York
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understanding... precedes development

The development of chlorothiazide and probenecid were events of major importance, but perhaps even more important for the future was the Renal Research Program by which they were developed. When Merck Sharp & Dohme organized this program in 1943, it was expressing in action some of its basic beliefs about research:

• Many problems connected with renal structure and function were still undefined or unsolved. The Renal Research Program would begin its basic research in some of these problem areas.

• From knowledge thus acquired might come clues to the development of new therapeutic agents of significant value to the physician.

For example, the Renal Research Program put fifteen years into this search before chlorothiazide became available. But because these years had first led to a greater understanding of basic problems, the desired criteria for chlorothiazide existed before the drug was developed.

Along with other research teams at Merck Sharp & Dohme, the Renal Research Program continues to add new understanding of basic problems—understanding which will lead to important new therapeutic agents.

MERCK SHARP & DOHME Division of Merck & Co., Inc., West Point, Pa.

where today's theory is tomorrow's therapy



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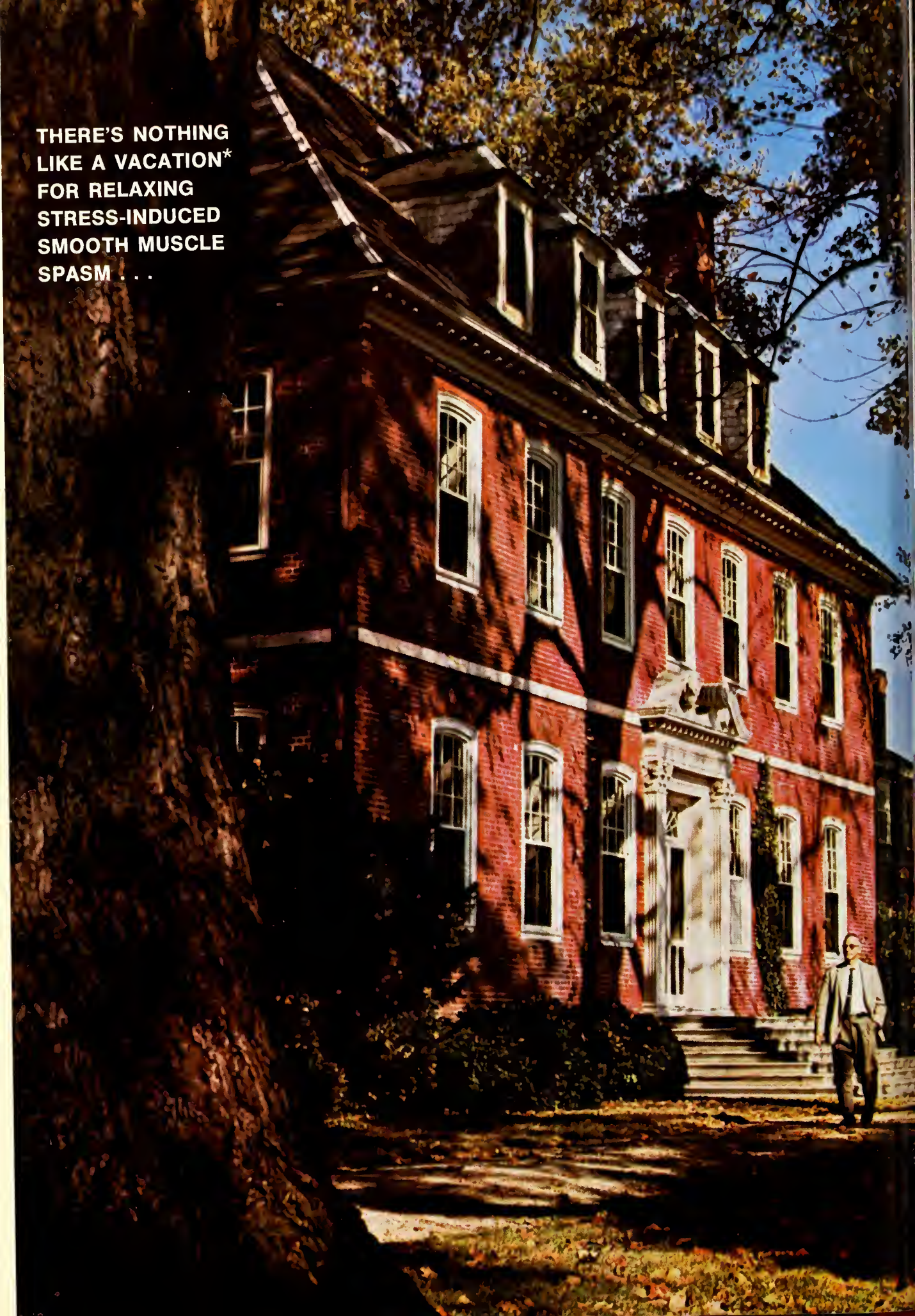
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Each capsule contains: Thiamine mononitrate (B_1), 15 mg.; Riboflavin (B_2), 10 mg.; Pyridoxine hydrochloride (B_6), 5 mg.; Nicotinamide, 50 mg.; Calcium pantothenate, 10 mg.; Ascorbic acid (vitamin C), 300 mg.

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SPASM . . .

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EXCEPT THE SEDATIVE-ANTISPASMODIC
BENEFITS OF

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There's nothing quite like a vacation to ease the pressures of the modern, "workingday" world. And for the patient who can't get away from it all, there's nothing quite like Donnatal to relax stress-induced smooth muscle spasm. For 31 years it has been the antispasmodic-sedative most often prescribed for relieving functional disturbances of tone and motility of the gastrointestinal tract.

belladonna alkaloids in optimally balanced ratio

In Donnatal, natural belladonna alkaloids are rationally balanced in a specific, fixed ratio that provides "the greatest efficacy with the smallest possible dose."[1] They avoid the clinical uncertainties of the variable tincture and extract of belladonna, and are considered superior in range of action to atropine alone.[2] Furthermore, they are generally recognized as being more effective than the synthetics for relieving visceral spasm.

phenobarbital for sedation

Years of clinical use have established phenobarbital as one of the most efficient and highly regarded sedatives. In fact, for general sedation it is the drug of choice.[3] In Donnatal, phenobarbital potentiates the spasmolytic effects of the belladonna alkaloids, lessening emotional tensions and checking the neurogenic impulses that trigger GI disorders.

more than 24 indications in PDR

Donnatal has withstood the test of time to become the classic sedative-antispasmodic because of its unsurpassed effectiveness, safety, economy, uniformity of composition, and dosage convenience. Its widespread acceptance and usage by the profession can also be attributed to its versatility in treating disorders characterized by smooth muscle spasm. There are more than two dozen distinct and separate indications for Donnatal listed in the current PDR.

IN EACH TABLET, CAPSULE, OR (5 cc.) OF ELIXIR

hyoscyamine sulfate	0.1037 mg.
atropine sulfate	0.0194 mg.
hyoscine hydrobromide	0.0065 mg.
phenobarbital	(1/4 gr.) 16.2 mg.

(warning: may be habit forming)

IN EACH EXTENTAB

hyoscyamine sulfate	0.3111 mg.
atropine sulfate	0.0582 mg.
hyoscine hydrobromide	0.0195 mg.
phenobarbital	(3/4 gr.) 48.6 mg.

(warning: may be habit forming)

BRIEF SUMMARY: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Administer with caution to patients with incipient glaucoma, or urinary bladder neck obstruction. Contraindicated in acute glaucoma, advanced renal or hepatic disease, or a hypersensitivity to any of the ingredients.

REFERENCES: 1. Vollmer, H.: Arch. Neurol. and Psychiat., 43:1057, 1940. 2. Morrissey, J.H.: J. Urology, 57:635, 1947. 3. Krantz, J.C., Jr., and Carr, C.J.: Pharmacological Principles of Medical Practice, 2nd ed., Baltimore (1954), 552.

*This one at Westover, elegant Colonial Virginia plantation, located on the James River near Richmond. Built in the early 1730's by William Byrd II, founder of Richmond, it is now the home of Mrs. Bruce Crane Fisher.

A. H. ROBINS COMPANY, INC., RICHMOND, VA.

A·H·ROBINS

AMA Retracts Statement . . .

THE AMA NEWS • JANUARY 31, 1966

Action Against Specific Machine, Not Against Diapulse Corporation

The federal government has filed a libel action in the U.S District Court for the Northern District of Georgia against a specific Diapulse machine seized in the office of an Atlanta chiropractor.

In an earlier story of the seizure of the machine, *The AMA News* said it was planned to file the action against the Diapulse Corp. of America, maker of the machine (*The AMA News*, Jan 10, 1966). The action is filed against the specific machine. There are no charges against the Diapulse Corporation nor against the manufacturer, Remington Rand Division, Sperry Rand Corporation.

The AMA News story also reported the fact that the American Medical Association's Dept. of Investigation said that some time ago representatives of the Diapulse firm had submitted data to an AMA department. In turn, the material was submitted to several outside consultants, who responded that the material was not persuasive that the device would do what was claimed for it in the reprints.

Jesse Ross, president of the Diapulse Corp. of America, said: "The Diapulse machine has been submitted for evaluation to universities, hospitals, and qualified doctors. The published reports which have appeared in medical journals have been very favorable to our product, including the areas of wound healing and infection."

- The American Medical Association, Chicago, on Dec. 19, 1965, stated to the Associated Press that it had "never seen or tested the device."
- At this same time, the US Naval Base Hospital at Oakland, California was using multiple Diapulse therapy units on 24 hour duty to treat Viet Nam casualties for infections and accelerating wound healing.
- The American Medical Association did not "consult" this or any other user of Diapulse.
- Thousands of users of Diapulse including private medical practitioners as well as other users in medical schools and hospitals are treating beneficially each day many thousands of patients with Diapulse therapy.
- Prominent Medical Journals have published the controlled and double blind studies using Diapulse therapy in qualified medical schools.

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Principal effectiveness as:
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Principal cautions and side effects: Use with caution in patients hypersensitive to sympathomimetics or barbiturates and in coronary or cardiovascular disease or severe hypertension. Insomnia, excitability and increased motor activity are infrequent and ordinarily mild.

Before prescribing, see SK&F product Prescribing Information.

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Blueprint for dealing with tension due to stress — Prolixin — once-a-day

For the patient who must be on the job mentally as well as physically, prescribe Prolixin. The prolonged tranquilizing action of as little as one or two mg. helps him cope with tension all day long. Markedly low in toxicity and virtually free from usual sedative effects, Prolixin is effective in controlling both anxiety associated with somatic disorders and anxiety due to environmental or emotional stress. Patient acceptance is good — because Prolixin is low in cost, low in dosage and low in sedative activity. Prescribe Prolixin.



Side Effects, Precautions, Contraindications: As used for anxiety and tension, side effects are unlikely. Reversible extrapyramidal reactions may develop occasionally. In higher doses for psychotic disorders, patients may experience excessive drowsiness, visual blurring, dizziness, insomnia (rare), allergic skin reactions, nausea, anorexia, salivation, edema, perspiration, dry mouth, polyuria, hypotension. Jaundice has been exceedingly rare. Photosensitivity has not been reported. Blood dyscrasias occur with phenothiazines; routine blood counts are recommended. If symptoms of upper respiratory infection occur, discontinue the drug and institute appropriate treatment. Do not use epinephrine for hypotension which may appear in patients on large doses undergoing surgery. Effects of atropine may be potentiated. Do not use with high doses of hypnotics or in patients with subcortical brain damage. Use cautiously in convulsive disorders. **Available:** 1 mg. tablets. Bottles of 50 and 500. For full information, see Product Brief.

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Stress formula vitamins are a key factor in bone and tissue regeneration. To meet the increased metabolic demands, STRESSCAPS offers therapeutic amounts of B and C vitamins as an aid to smoother convalescence and earlier rehabilitation. In fractures, as in many other conditions of physiologic stress, STRESSCAPS vitamins are therapy.

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Vitamin B_1 (Thiamine Mononitrate)	10 mg.
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Miltown® (meprobamate)

Indications: 'Miltown' (meprobamate) is effective in relief of anxiety and tension states. Also as adjunctive therapy when anxiety may be a causative or otherwise disturbing factor. Although not a hypnotic, 'Miltown' fosters normal sleep through both its anti-anxiety and muscle-relaxant properties.

Contraindications: Previous allergic or idiosyncratic reactions to meprobamate or meprobamate-containing drugs.

Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

Supplied: In two strengths: 400 mg. scored tablets and 200 mg. coated tablets.

Before prescribing, consult package circular.

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, V., and Dawson, A.: l. J. *1*:293, 1965.



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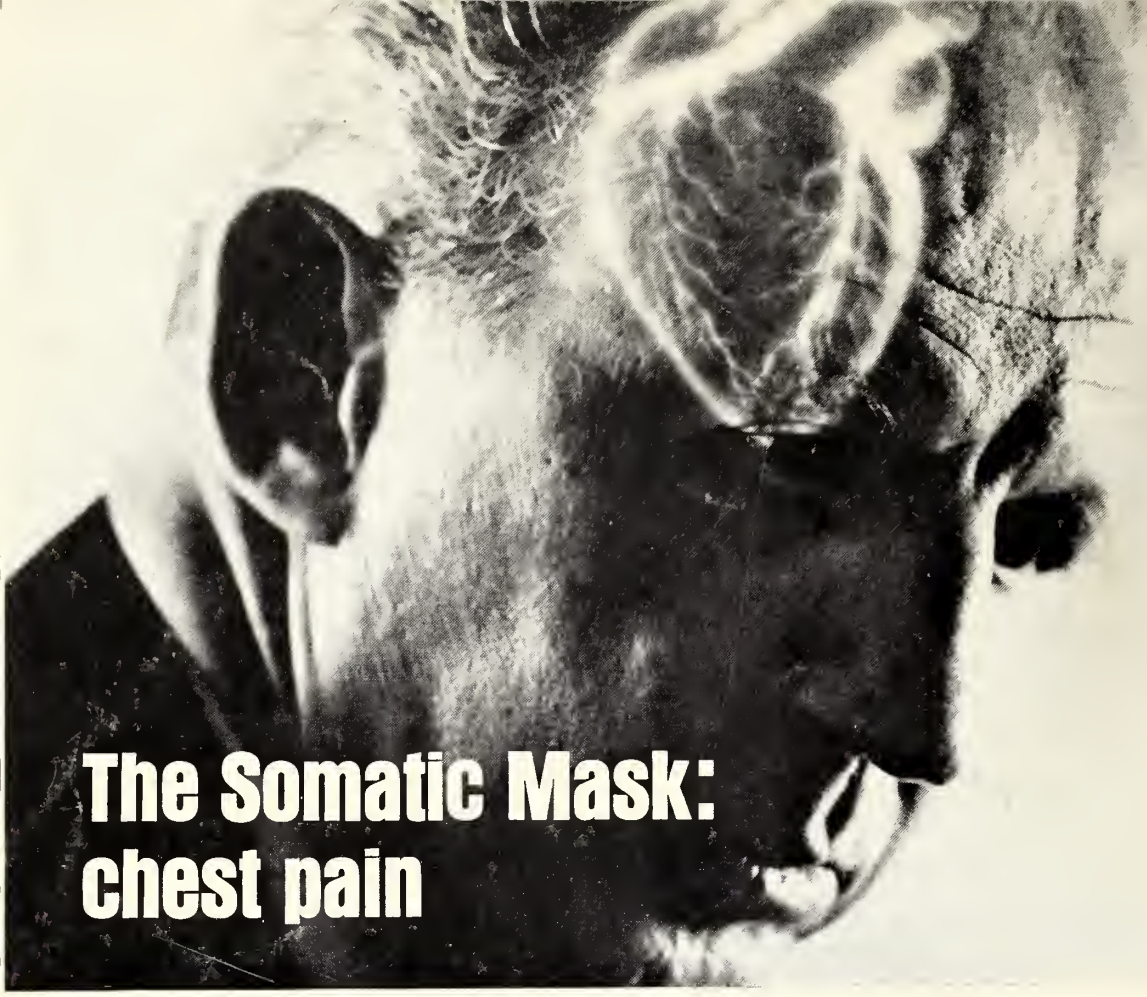
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Valium (diazepam) is generally well tolerated, and usually does not impair mental acuity or ability to function. If side effects such as ataxia and drowsiness occur, they usually disappear with dosage adjustment.

In prescribing: Dosage—Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hrs, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

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PRECAUTIONS: Periodic examination of the blood is advisable. Nystagmus in combination with diplopia and ataxia indicates dosage should be reduced.

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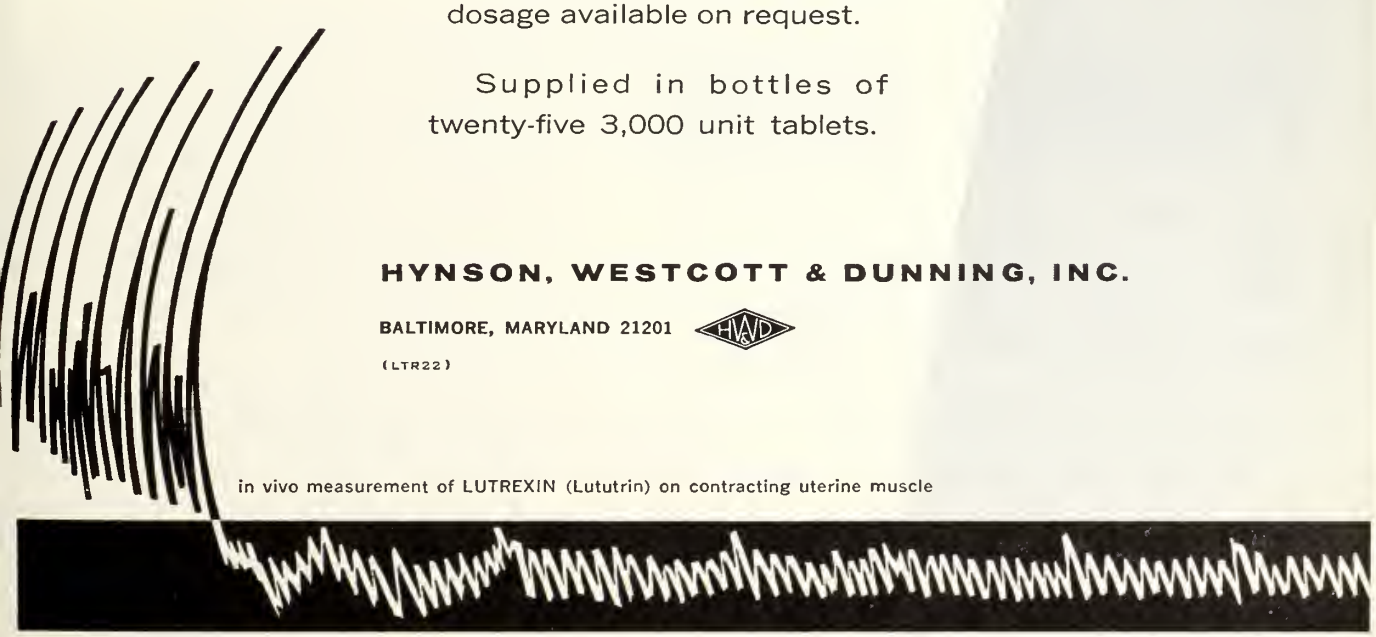
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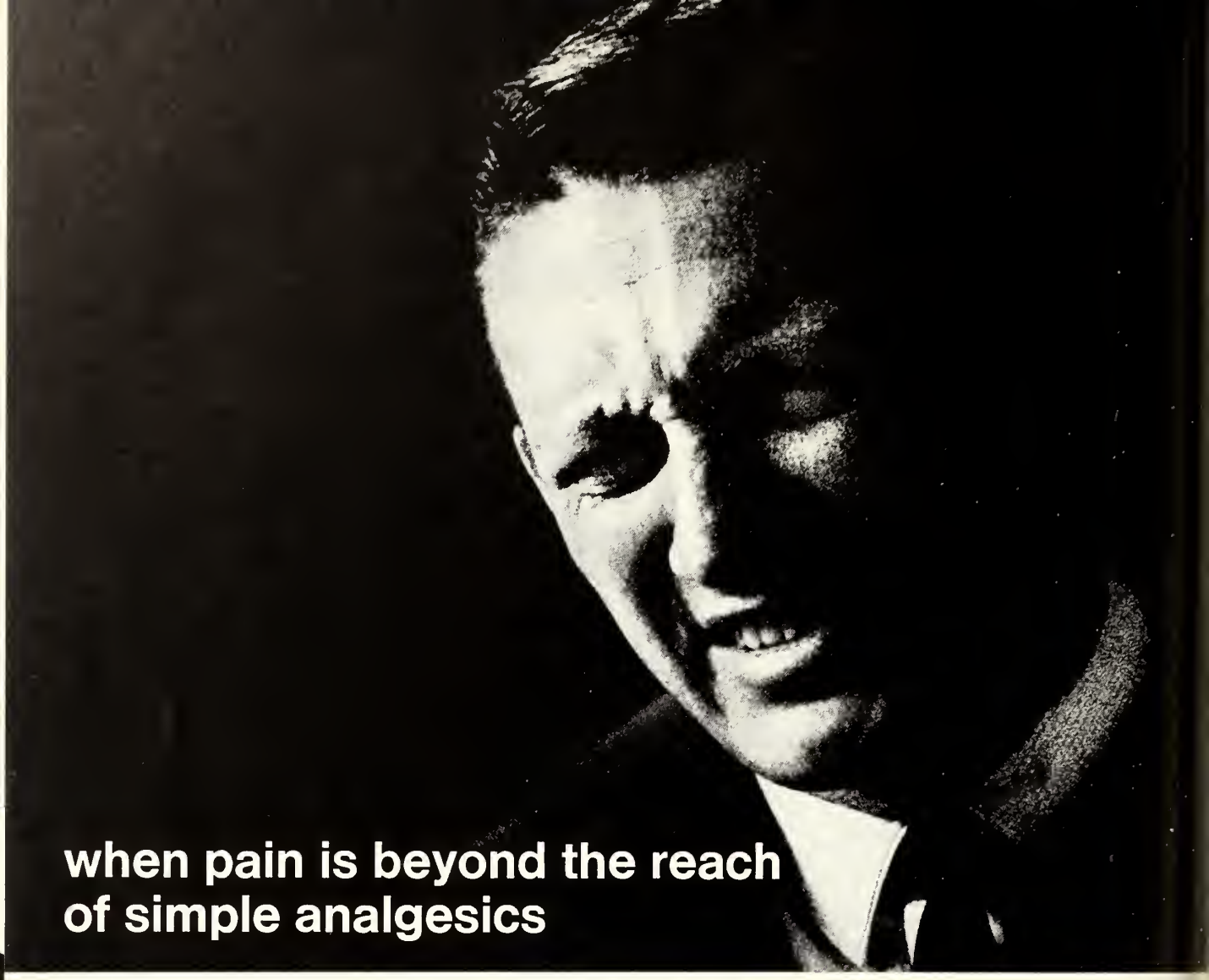
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1. Collective studies, Department of Medical Research, Winthrop Laboratories.

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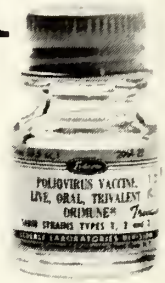
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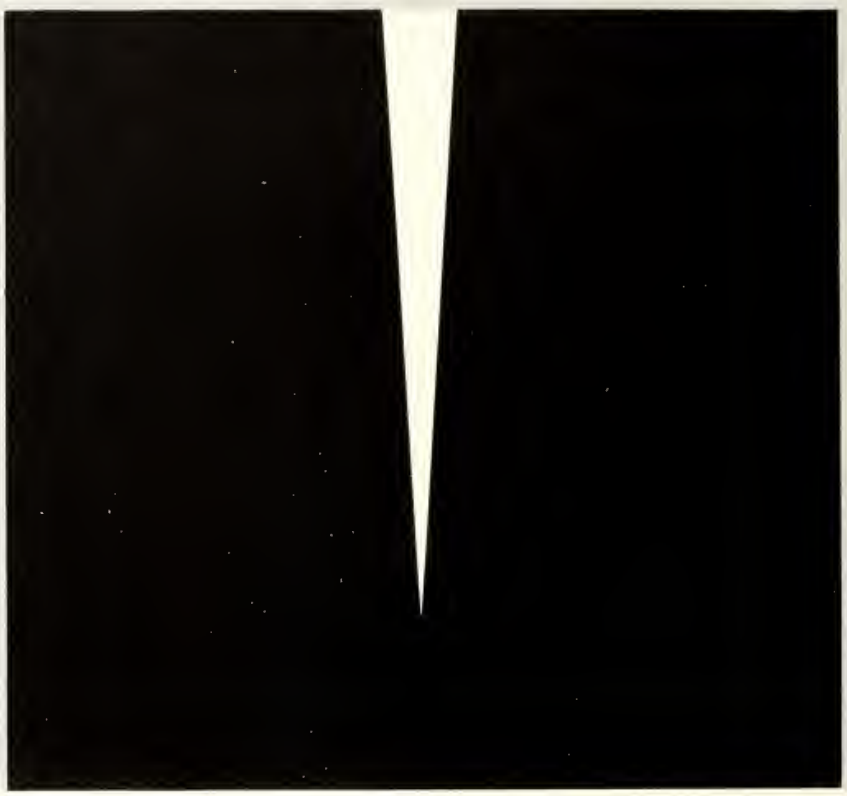
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Loosening epithelial cells of nasal mucosa during early stage of cold—Approx. 1800× Magnification.

this issue: partners in misery

Partners in misery: common cold and duodenal ulcer*



A century or two ago our forefathers believed that the common cold was caused by an excess of evil humors. Today we regard these baleful humors as a result of the cold rather than its cause. Only in the last ten years have some of the offending viruses been cultivated, and we now know many others can be transmitted to human volunteers although they cannot yet be grown in the laboratory. Various body defenses react to infection by a virus, and in the respiratory tract the lung is protected by the phagocytic properties of the pulmonary alveolar macrophage.[1]

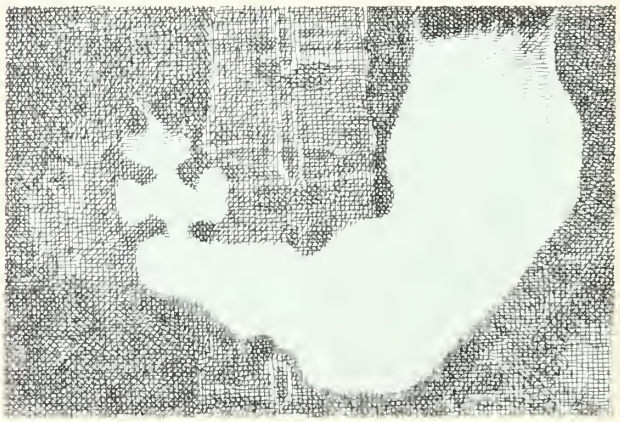
Because of the brief history and large proportion of unidentified viruses, effective prophylactic vaccines have not yet appeared. A universal antiviral agent seems even more remote. Meanwhile, these viruses continue to produce epidemics of upper respiratory infection at seasonal intervals, particularly in spring, fall and winter, during which they propagate and distribute progeny indiscriminately. As a result, they are responsible for significant work loss. Since the immunity afforded is temporary, recurrent infections are common.

This recurrent morbidity in spring and fall is shared by duodenal ulcer, itself a cause of significant loss in wages and medical expense. Thirty years ago Emery and Monroe reported 1,279 recurrences of peptic ulcer of which 13 per cent could be attributed to upper respiratory infection, thus confirming a long-held clinical impression.[2] While the inflammation, congestion and secretion of the mucosa of the nasopharynx in the common cold, and the hyperemia and hypersecretion of the gastric mucosa during acute exacerbation of a duodenal ulcer may not be directly linked, it is probable that the miseries of the infection are reflected in the pain of the ulcer. The following case history illustrates this point.

Case report A 48 year old white male executive, suffering from hematemesis and melena for 12 hours was admitted to hospital on April 8, 1965. He gave a history of duodenal ulcer proved by x-ray studies 23 years previously. In the intervening years he suffered from typical epigastric pain occurring two hours postprandially, usually for two or three weeks each spring and fall. A hospital admission two years earlier had followed hematemesis of moderate amount.

The present admission was preceded by a recurrence of pain during a period of unusual business pressure. In addition he caught a cold one week before his entry to hospital and his already irregular eating habits were made more so by loss of taste and appetite, blocked nose and general malaise. During the final 36 hours he used aspirin to relieve his cold, taking 2 or 3 tablets with a glass of water on 6 or 7 occasions. Before retiring he drank a glass of hot toddy.

He awakened about 1 a.m. and vomited dark red blood and recently ingested food mixed with whisky. Melena followed and he was transferred to hospital. On admission he was pale, cold and sweating. Apart from epigastric tenderness and black stool on the examining finger after rectal examination, his general physical examination was not remarkable. Temperature was normal, pulse was thin and thready with a rate of 114 beats per minute, and blood



The commonly found "clover-leaf" roentgenographic abnormality of chronic duodenal ulcer due to spastic contractions and scarring.



Typical site of duodenal ulcer showing anatomical relationship to gastroduodenal artery and common bile duct.

pressure measurement was 104/60mm. of mercury. Hemoglobin estimation was 8.6 grams per cent, but white blood count, chest x-ray and urinalysis were within normal limits. No further bleeding occurred. After blood transfusions and a suitable medical regime he was dismissed two weeks later and advised to return later for consideration of surgery.

The care of the ulcer patient suffering from a viral infection of the upper respiratory tract is additionally handicapped by the local and constitutional symptoms so produced. Indifference to food often makes it difficult to "feed a cold" even in a patient without an ulcer. In the ulcer patient this becomes more than a homily, for malaise and apathy towards irksome routine produce a lack of interest in adhering to an ulcer program and result in its eventual failure if these symptoms are not relieved. The absence of curative treatment for these viral infections has spawned many a strange therapeutic offspring and some of the more generally accepted remedies contain a special risk for the ulcer patient.

To the gastric physiologist, the shot heard around the world was not fired at Concord but was discharged through the stomach of Alexis St. Martin. The worthy voyageur had a more than modest thirst and on several occasions William Beaumont observed through the resulting fistula the changes of gastritis which followed an immoderate ingestion of alcohol.[3] It is now well known that alcohol stimulates the production of gastric juice and the patient with an ulcer, even in remission, should not drink alcohol without first taking food, milk or antacid. The use of various alcoholic remedies for the common cold is to be strongly discouraged in this type of patient. Factors already present can produce an acute exacerbation of ulcer symptoms without the added stimulus of alcohol.

Headache and facial pain, sore throat and generalized aching are subjective discomforts of viral infections of the upper respiratory tract or their complications. Relief from them is usually sought in the family medicine cabinet, and of all the medications habitually stored there, salicylates are the most frequently used. Oral ingestion of salicylates has produced



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substernal pyrosis and sometimes epigastric pain in susceptible individuals. Bleeding from erosions of the gastric mucosa has followed their use and has been severe enough in some instances to cause hematemesis and melena.[4] It has been suggested that interference with the protective mucous layer of the stomach allows this to happen. There is no evidence that hypersecretion occurs, but the taking of aspirin has been followed by exacerbation of ulcer symptoms and occasionally by gastrointestinal bleeding.[5] The ulcer patient with a cold should take salicylates with an antacid, or preferably other means of symptomatic relief should be found.

Caffeine has two actions on gastric secretion. One directly stimulates production of acid and the other potentiates the out-pouring of gastric juice as a response to other stimuli.[6] In high doses to animals, it has produced erosive gastritis and peptic ulceration. Caffeine-containing beverages are discouraged for the ulcer subject and forbidden during an acute exacerbation.[7] Many cold remedies contain caffeine.

To sufferers from allergic rhinitis springtime brings symptoms of nasal obstruction, loss of taste and smell and malaise similar to those caused by viral infection but due instead to allergens which at that time of year are principally tree pollens. Relief from these symptoms can be obtained, though not wisely, by the use of steroid medication. Unfortunately, chronic sufferers from these allergies have used this approach with varying degrees of success. Steroid hormones increase gastric secretion and delay the healing of experimental ulcers.[8,9] Clinically their administration has been associated with reactivation of healed duodenal ulcers, and with bleeding and perforation which were not always preceded by typical ulcer distress. Because of these harmful effects, their use in the ulcer patient is best avoided for other than serious medical problems and then only with adequate antacid coverage.

Summing up It is immaterial by which pathways the malaise and lassitude, depression and irritability activate an ulcer, but it is the physician's responsibility to ensure that the medications he selects to relieve the symptoms of a cold do not further aggravate the ulcer.

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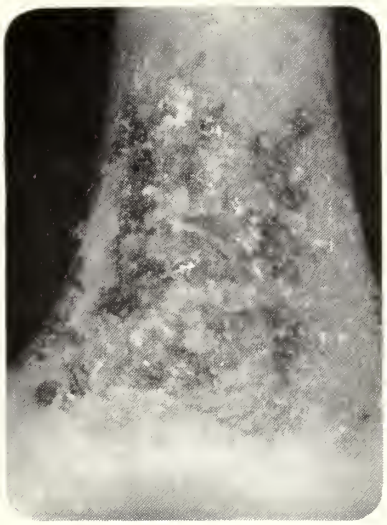
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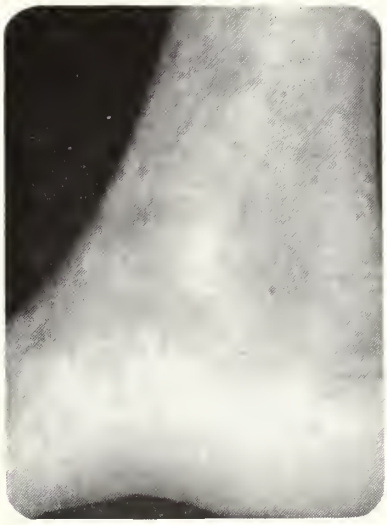
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Addison's Disease

A Case Report

Thomas F. Conneen, M.D.*

The adrenal insufficiency of Addison's Disease is a comparatively rare entity. The symptoms are those that might be encountered in general medical practice–asthenia, loss of appetite, loss of weight and G.I. disturbance. In the typically neurasthenic patient, Addison's Disease is often considered. Good clinical judgment is usually correct in the differential diagnosis. Often, confirmatory tests are necessary to disprove the presence of adrenal insufficiency rather than to prove it. With the current explosive expansion in adrenal physiology, the clinical picture has become extensive and complicated and frequently, for the non-specialist, confusing. The purpose of this paper is to present a case of Addison's Disease which was a diagnostic problem in its early stages and to discuss the clinical diagnosis and treatment of adrenal insufficiency.

Case Report

A 16-year-old junior in high school was seen in the Hospital on January 5, 1966 with the chief complaint of being tired since August of 1965, stomach trouble described as an intermittent ache, being easily filled after a small meal, constipation, nervousness, generalized weakness and weight loss. His history dates back to the Fall of 1964 when his mother noted that he seemed to be more fatigued than usual with a decrease in appetite and complaining of his stomach not feeling well. This, she noted, usually occurred following basketball practice.

In January, 1965 he had to drop basketball because of poor grades, following which his history professor who was also the coach of basketball and a personal friend of the family, gave him the 'cold shoulder'. In July of 1965 he had his first physical examination, at which time he weighed 107 lbs. The examination was said to have been negative. He was apparently tired during the summer; however, he ate well. In August he was helping his father on Saturdays and once fainted. His father described it as a fainting spell which he thought was due to heat prostration. In September he began the school year and after approximately two weeks, he complained of stomachache and feeling tired. He was examined by another doctor who noted nothing specific and he was admitted to a hospital, somewhat dehydrated, but no specific diagnosis was made after an extensive work-up.

After his discharge from the hospital in September, his teachers were consulted and he took only two subjects because of weakness and fatigue. He went to school for one to two weeks in November until he developed an ear infection which responded to antibiotics. Following this he was fairly well until just before Thanksgiving when he had "the Flu" associated with diarrhea and vomiting. Following his recovery from "the Flu" he was eating well and did fairly well until before Christmas when he began to complain again of stomachache and increasing fatigue, as well as generalized weakness. Several days before his admission to hospital, January 5, 1966, he fainted while straining at the stool.

His past history revealed that he had a tonsillectomy as a child and no other illnesses other than the ordinary childhood diseases.

His mother and father are alive and well; his mother had tuberculosis in 1944, six years before the child was born and was treated in Fairfield Sanitorium for eight months. After discharge she was readmitted five to six months later because of a recurrence. He had one sister aged 7 who was adopted and whose health was reported as good.

Social History: Revealed that the child was quiet and shy and described by some of his classmates as a 'loner'. He was a junior in high school and played the trumpet until this year. Approximately a year before admission he had to stop basketball because of poor ranks and fatigue following practice. He had trouble with his studies in high school and had to work real hard. However, in the eighth grade he was very successful and was first in his class, tied with two other students.

Physical Examination: Revealed a cooperative male whose skin was dark in color which he attributed to his residual tan from the previous summer. He was very thin; appeared weak and chronically ill, with listless and indistinct speech. Head–normal, eyes–pupils were equal and reactive to light and accommodation, extra ocular movements were normal, field of vision was normal, no nystagmus or scleral jaundice. Ears were normal. The mucous membrane of the mouth showed no definite areas of pigmentation; however, on close examination there was evidence of a pigmented line at the gingival border. Tongue was normal. Neck revealed no palpable adenopathy. The thyroid gland was not palpable. There were no supraclav-

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icular or axilliary lymph nodes. Heart revealed normal sinus rhythm, no cardiomegaly or murmurs. Lungs were clear to auscultation and percussion. Abdomen was soft without distention. Bowel sounds were active. Liver, kidney and spleen were not palpable. Genitalia, negative. Rectal examination was negative with the exception of perianal pigmentation. Extremities were thin, reflexes were active, peripheral pulses were palpable. There was no evidence of peripheral edema. Extensor plantor responses were normal. Sensation was normal. Skin showed generalized pigmentation including the area covered by the bathing suit, with pigmentation of scars and also perianal pigmentation as well as increased pigmentation over pressure areas particularly over the scaral area, buttocks, knees and elbows.

Laboratory Investigations: Revealed a small heart, skull X-ray was negative, without evidence of enlargement of the pituitary fossa or any erosion of sella tursica. A KUB did not reveal adrenal calcification seen frequently in tuberculosis. The hemoglobin was 15.2 gms. hematocrit 46, WBC normal with a normal differential count, sedimentation rate 3, total eosinophil count 88; urinalysis negative, urine for culture and sensitivity revealed no growth, urine for acid fast culture was negative, and the Tine test was negative. BUN 30, FBS 92, sodium 129, potassium 5.2, CO_2 25, chloride 98, cholesterol 88, stool showed a trace of occult blood, blood sugar, one hour PC. 108; T3 uptake was 32%; urine 24 hour sodium 67 mEq., baseline 17-hydroxycorticoids revealed 1.0 mgm. per 24 hours; baseline urinary 17-ketosteroids 1.10 mgm. per 24 hours. Repeat, following stimulation with ACTH intravenously over 8 hours, revealed 17-ketosteroids 1.3 and 17-hydroxysteroids 2.1. Second day following stimulation with intravenous ACTH over 8 hours, revealed 24-hour urinary 17-ketos. 2.3, 17-hydroxysteroids 2.2.

Discussion

The clinical history, without seeing the patient suggested neurasthenia or psychoneuroses in a young boy. However, the patient on clinical examination gave the appearance of having Addison's Disease. Laboratory investigations were consistent and confirmary of Addison's Disease. The adrenal insufficiency of Addison's Disease is often easily recognized from physical examination and history. Laboratory aids are useful in confirming the diagnosis, especially in borderline cases. Before frank Addison's Disease occurs, a period of limited adrenal reserve must always be present in which the hormone is manufactured in sufficient amounts for ordinary activity, at which time a purely clinical diagnosis may be difficult or impossible without appropriate laboratory studies. However, if undiagnosed in the early stage will progress to progressive weakness, loss of weight, complete disability and eventually death. The above patient was treated with cortisone acetate 25 mgs. and Florinef® 0.1 mg. each morning with breakfast. While in hospital on this program, he felt immediately 100% better and gained weight. His low weight in hospital was 82 lbs. and on discharge his weight was 94 lbs.

ADDISON'S DISEASE is not common. The Mayo Clinic's incidence is 16 per 100,000 patients. There is no particular sex predilection. The highest age incidence is between 30 and 50 years.

The onset is insidious usually. Rarely an adrenal crisis in a stressful situation is the first manifestation. Weakness is present in 99% of the cases, pigmentation of the skin occurs in 98% of the cases, pigmentation of the mucous membrane in 82%, weight loss is a very prominent feature without which a definite diagnosis of Addison's Disease cannot be entertained. This occurs in 97% of cases. Anorexia, nausea and vomiting is frequent and present in approximately 90% of cases. Hypotension below 110/70 and small heart size is found in approximately 87% of cases. Symptoms of abdominal pain, salt craving, diarrhea, syncopy and vitiligo are less commonly found. Occasionally one may see hypoglycemic manifestations. There may be nervousness and other mental symptoms and in long standing disease the speech is listless and indistinct.

It was always thought that weight loss in Addison's Disease was the result of dehydration and anorexia. However, McBrien et al. from England, emphasize steatorrhea as the main cause. They studied four Addisonians and each one had steatorrhea that subsided after treatment of their disease with steroids. One should stress that gonadal function and secondary sex characteristics are rarely disturbed. The menses in the female patient are normal. The pigmentation in Addison's Disease is primarily the result of excessive corticotropin and melanin stimulating hormones produced by the pituitary and only secondarily the result of adrenocortical insufficiency. There have been two reports of Addison's Disease without abnormal pigmentation. However, the pigmentation is a cardinal manifestation of chronic adrenal insufficiency. Diabetes mellitus and Addison's Disease is being reported more frequently. To this day there are 81 patients reported. In the majority of cases the diabetes came first. In diabetic patients, where a marked decrease in insulin requirement is seen and where severe hypoglycemic reactions are encountered, one should look for coexistent Addison's Disease.

The etiology of Addison's Disease until recently, was generally conceded to be due to tuberculosis in 80 to 90% of the cases and atrophy for the vast majority of the remainder. In recent years this ratio appears to be changing with atrophy accounting for more than 60% of the cases, the cause of atrophy being unknown. Other causes of adrenal insufficiency include fungal infections; coccidioidomycosis, histoplasmosis and blastomycosis. Metastastic carcinoma is being seen more frequently as is iatrogenic adrenal insufficiency due to abrupt withdrawal of corticosteroids in the treatment of various medical diseases. Surgical removal for palliation of metastatic breast carcinoma following oophorectomy is another cause of Addison's Disease. Less common are such conditions as primary disease of the anterior pituitary, amyloidosis and hemochromatosis. Lately, some evidence has been brought forth to advance the concept that Addison's Disease is of auto-immune origin. Blizzard, et al., found circulating antibodies to the adrenals in 16 out of 30 Addisonians.

The diagnosis of Addison's Disease or insufficiency of the adrenal cortex is made by history, physical examination and confirmed by specific hormone measurements.

Methods are now readily available for direct measurement of the blood or urinary levels of adrenal steroids. These should be employed whenever possible and certainly before a definitive diagnosis of adrenocortical insufficiency is established. Baseline values of blood and urinary steroids are useful; however, it is apparent that the baseline values in early cases of adrenal insufficiency may still lie within the normal range when reserve function or the capacity of the gland to respond to stress is markedly impaired. Thus a presumptive diagnosis of early adrenal insufficiency is made certain on the basis of blood or urinary steroid response to specific stimulation of ACTH. I have found the most satisfactory way to make a definitive diagnosis is to collect a 24-hour urine for 17-hydroxycorticosteroids and 17-ketosteroids on the first day as a baseline. The following two days, repeat 24-hour urine samples for 17-hydroxy and ketosteroids are obtained. However, on the second and third day from 7 a.m. to 3 p.m. an intravenous of 500 cc. of normal saline containing 20 to 50 units of ACTH is infused. The amount of ACTH is not the important factor, it is the duration of the intravenous infusion. It is not absolutely necessary that the result of the 24-hour urine 17-hydroxycorticosteroid be obtained. In the normal individual there is a rise from approximately 4.5 17-hydroxycorticosteroids in mgm, per 24-hours to 18 following ACTH stimulation, whereas in a patient with Addison's Disease the baseline may be in the range of 1.9 without any significant elevation following stimulation with ACTH.

In normal subjects, therefore, there is an increase of at least 7-15 mgms. in the 24-hour collection for urinary ketosteroids and 17-hydroxysteroids following ACTH stimulation over 8 hours. In Addison's Disease the response is subnormal. If poor adrenal reserve is suspected then additional ACTH stimulation may be necessary on subsequent days. Some people prefer to measure plasma corticosteroids after I.V. ACTH. They rise from a normal baseline level of 15 micrograms per 100 mml. to 40 micrograms or more. In Addison's Disease the rise is up to 20 micrograms and certainly no higher.

Normally the sodium potassium ratio is in the range of 30. In Addison's Disease, it falls towards 20. However, in cases of early Addison's Disease in which a patient maintains a high salt intake, secondary to salt craving, the serum sodium and potassium may be normal. The 4-hour ACTH test was at one time considered most useful for clinical evaluation of adrenal function. It depends upon the effect of adrenal cortical steroids in diminishing the number of eosinophils in the peripheral circulation. In individuals with normal adrenal function, an i.m. injection of 25 mgs. of ACTH produces a fall of 50% or more in the blood eosinophil count at the end of four hours. However, this response is not pathognomonic of adrenal inadequacy; it occurs also in certain cases of allergy.

When the suspicion of adrenal insufficiency is low, the water tolerance test may be used. 1500 cc. of water, (about 20 ml. of body weight) is given to the fasting patient and urine collections are made with the patient in a recumbent position. If there is less than 1000 ml. of urine output, impaired water diuresis is suspected. If there is diuresis after 50 mgm. of cortisone or hydrocortisone given orally or parenterally, adrenocortical insufficiency is strongly suggested.

A rapid test for adrenocortical insufficiency is reported by Maynard et al., in the March 1966 issue of the *Annals of Internal Medicine*. This report describes a rapid and efficient method for differentiating normal adrenal function from adrenal insufficiency, utilizing the acid fluorescence determination. A single intramuscular injection of 25 units aqueous ACTH is injected to stimulate the adrenal glands. One 8-10 ml. blood sample is obtained in a heparinized tube immediately prior to the injection of ACTH and one hour after ACTH. The normal plasma acid fluorescent steroid level measuring the baseline is between 6.3 and 35 micrograms per hundred ml. One hour following ACTH stimulation the plasma acid fluorescent steroid level in a normal individual rises to 11.3-47.8 micrograms per hundred ml. In a patient with Addison's Disease there is a rise to a maximum of 2.6 micrograms per hundred ml. This particular means of differentiating the normal adrenal function from adrenal insufficiency is felt to have two advantages. First, specimen collection requires little time and since no special preparation is necessary, it is applicable to out-patient testing. Second, the fluorometric method for cortisol determination is rapid and relatively specific.

During the ACTH tests one should protect the patient by giving 1 mgm. of dexamethazone or 9 alfa-fluorohydrocortisone 0.1 mgs. orally, one hour prior to the test to avoid anaphylactic reaction as a result of the foreign protein in ACTH in sensitive individuals lacking hydrocortisone protection. This will not interfere with the 24-hour urine collections for steroid metabolites.

The treatment of Addison's Disease is directed towards replacement of the deficient steroids approximating as closely as possible the steroid secretion by the adrenal gland in the normal individual. Experimental and clinical evidence indicates that practically all the clinical and metabolic features of the disease depend on decreased function of the adrenal cortex alone. The essential features of the clinical picture of Addison's Disease can be produced experimentally by adrenalectomy and adrenalectomized animals can be maintained in a normal state by administration of extracts of the adrenal cortex. For clinical and physiological purposes it is sufficient to consider that the adrenal cortex secretes three general classes of hormones. These are the glucocorticoids, the mineralocorticoids and hormones possessing androgenic or estrogenic activity. Although numerous steroids have been isolated from the adrenal cortex only a few appear to be of metabolic significance. The predominant steroids in man are hydrocortisone,

altosterone, androgen and estrogen. Since the normal individual secretes approximately 20 mgm. of hydrocortisone and 0.1 mgm. aldosterone per day, as the primary glucorticoid and mineral corticoid respectively, replacement of these hormones by steroids with similar metabolic activity is indicated. A simple method advised by Cahill & Thorn is cortisone acetate, 25 to 37.5 mgm. and 9-alpha fluoro-hydrocortisone acetate 0.1 mgm. daily, orally. They prefer to give 25 mgm. of cortisone and 0.1 mgm. of Florinef (9-alpha-fluorohydrocortisone) in the morning and 12.5 mgm. of cortisone acetate in the evening. These are never taken on an empty stomach and are best taken with meals. The best guide for adequate therapy is the patient's sense of well being. An objective and secondary guide is the patient's pigmentation. If there is a loss of appetite and weakness, an increased dosage should be anticipated. However, any patient who requires more than 37.6 mgm. of cortisone daily, probably has some other problem and any increase in steroids above this amount except for brief periods of stress or similar circumstances should be carefully re-evaluated. Patients with diabetes, hypertension and neurotic or phychotic disorders should be regulated on a lower dose of glucocorticosteroids if possible. Likewise, elderly patients or patients with hypertension or heart, renal or liver disease, mineralocorticoids may either be tapered to 0.05 mgm. daily and in some patients omitted completely. Steroid administration should be increased during times of stress such as during major surgical operations, trauma, obstetrical delivery, upper respiratory tract infection and dental extractions and gradually reduced back to maintenance dose in two or three days. This also applies to patients who have been on prolonged steroid therapy or on steroids in the year prior to major surgical operations. These patients should have the so-called "steroid prep" before, during and after surgery.

In previous years most patients received tri-weekly or monthly injections of deoxycorticosterone mineralocortical replacement. However, thanks to the ease of administration of the oral fluorohydrocortisone and even more precise daily administration compared to fluctuating levels of the depo preparations, most patients have been switched to oral medication.

The treatment as outlined above usually results in striking clinical and metabolic improvement with increase in weight, plasma volume and restoration of normal homeostasis with improved strength and sense of well being.

The natural course of the disease until the recent introduction of specific therapy was usually progressively downward with acute exacerbations at irregular intervals. Prior to 1930 the five-year mortality was 80% at which time there was no specific treatment. Between 1930 and 1937 when patients were treated with high sodium, low potassium diets the five-year mortality was in the range of 75%. In 1937 the synthesis of the first natural corticosteroid was achieved, namely deoxycorticosterone. Between 1938 and 1948 the five-year mortality was 50%. Between the years 1950-55 with the advent of both cortisone and hydrocortisone, in conjunction with use of deoxycorticosterone, the mortality was significantly reduced to approximately 15%. With the introduction of improved methods of treatment the prognosis of Addison's Disease has altered considerably and in many cases not only is life saved, but the patient is able to resume his normal activities. He can withstand surgical operations and intercurrent infections.

Before concluding, one should be made aware that acute adrenal crisis may occur without recognizable cause or following dietary indiscretion, intercurrent infection or trauma of any nature, including surgical operations. The incidence is highest with the onset of hot weather. The earliest symptoms of adrenal crisis are anorexia, vomiting, epigastric discomfort with increasing asthenia and lethargy. The extremities may be cold and temperature subnormal with associated fall in blood pressure and coma develops within a few hours. Death occurs unless adequate treatment is instituted promptly with steroids and saline intravenously.

Conclusion

The patient with extreme pigmentation and marked wasting is now a rarity in clinical practice due to earlier and more astute diagnosis by the medical profession of Addison's Disease. The challenge of early diagnosis, however, is great, since the mild weakness, slight anorexia and ill-defined malaise of mild Addison's Disease makes this patient more difficult to separate from the vast population of patients with these complaints due to other pathological or functional disorders.

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Emphysematous Bullae with Air-Fluid Levels Associated with Undiagnosed Pulmonary Tuberculosis

A Case Report Including Autopsy Findings

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INTRODUCTION

The development of emphysematous bullae in association with active pulmonary tuberculosis is not an unusual clinical finding; however, rarely have preexisting bullae become infected by tuberculosis.[2] In the ten reported cases found in the literature, the diagnosis of tuberculosis bullae was facilitated either by a history of previous pulmonary tuberculosis, or by the finding of subsequent acid fast organisms in the sputum, or by radiologic evidence of tuberculous disease in adjacent lung tissue.

The present case report of an infected emphysematous bullae is interesting in that tuberculosis was undiagnosed during the patients' lifetime. Only at postmortem examination was the etiology of the infection determined.

CASE REPORT

A 79-year-old white retired insurance salesman was admitted to the medical service of an out of state hospital on January 14, 1963 in severe respiratory distress. He had been followed in the medical out-patient service since June, 1962 with the diagnosis of pulmonary emphysema. His clinic record showed that he had chronic bronchitis throughout his adult life and had smoked between one and two packs of cigarettes daily until 1950, having stopped his smoking at that time because of increasing dyspnea. In 1952, he was hospitalized for a spontaneous pneumothorax of the left lung from which he made a satisfactory recovery. In spite of progressive dyspnea, he remained ambulatory until September, 1961, when he was admitted to another hospital for an acute back injury. While there, emphysema with severe pulmonary insufficiency was noted. The roentgenogram of the chest showed bilateral pulmonary bullous formation and pneumonic processes involving the left upper and right lower lobes of the lung. Satisfactory improvement was made while on penicillin and nebulization therapy. He was discharged after two months hospitalization and referred to the medical clinic for follow up care of his pulmonary disease. In June, 1962 weakness and severe dyspnea was noted by the patient. The roentgenogram of the chest showed the previously described bullous formation of the lung without evidence of an inflammatory process. (See Fig. 1) His symptoms improved while on expectorants, a mycolytic agent and nebulization therapy with bronchodilators. He remained active until his illness in December, 1962 when he developed severe dyspnea, profound weakness, cyanosis, and a cough productive of a slight amount of grayish, watery sputum. All these symptoms were evident at the time of his hospital admission three weeks later. In the past, he had not experienced fever, chills, night sweats or hemoptysis. He had no history of allergies, asthma or tuberculosis.

Physical findings on admission revealed a tall, thin, cachetic elderly white male in acute distress with dyspnea on exertion and on phonation. Blood pressure 110/70; pulse 110/m.; respiration 36/m.; temperature 98.6 orally. Cyanosis was present about the lips and fingertips. There was grade one retinopathy (Keith-Wagner) without papilledema. The accessory respiratory muscles of the neck and upper thorax were hypertrophied. The chest moved en masse with respirations and an increased anteroposterior diameter of the thorax was noted. The lungs were hyperresonant to percussion. On auscultation there was noted prolongation of the expiratory phase of respiration; diminished breath sounds, especially beneath the right scapula; scattered inspiratory rales and a sucussion splash below the right scapula. Cardiomegaly was thought to be present with the point of maximal impulse at the sixth intercostal space and anterior axillary line. Tachycardia with an apical systolic gallop was present. The pulmonic second sound was increased and a grade two ejection murmur was heard at the apex. The abdomen was soft, non tender, the abdominal aorta was enlarged to the right of the midline. No organomegaly was noted. Moderate muscle wasting of the extremities and clubbing of the fingers were observed.

Laboratory Studies: White blood cell count 10,800 with 84 segmented, 2 bands, 8 lymphocytes, 2 sosinophils, 4 monocytes. Hemoglobin 15, hemotochrit 46. Blood urea nitrogen 22 mg/%; fasting blood sugar 80 mg/%; blood carbon dioxide 26.4 meq./liter; blood serology nonreactive; arterial oxygen partial pressure 51 mm.; and carbon dioxide partial pressure 52 mm; arterial Ph 7.43; three blood cultures were negative after 14 days; two sputum cultures showed normal flora, and one sputum was negative for acid fast bacillus. Tuberculin skin test first strength was negative in 48 hours. Electrocardiogram showed a vertical axis with deep S wave in V6 and a small $S\text{-}R^1$ in V1 and V2, suggestive of pulmonary hypertensive heart disease. Radiologically, the right lung contained a large air cyst occupting the region of the right upper lobe which showed multiple air fluid levels filling approximately one-third of the cavity. The heart and mediastinal structures were displaced across the midline to the left. Compression atelectasis of the remainder of the right lung and the left upper lung was present. (See Fig. 2)

Clinical Course: The impression was that pulmonary insufficiency with hypoventilation and compensatory respiratory acidosis had developed as a result of a superimposed infectious process upon severe bullous emphysema. The patient was treated with intermittent positive pressure breathing, nebulization with bronchodilators, iodide solution, tetracylines and digitalis. On this medical management, clinical improvement was noted over a two-week period. His temperature, taken every six hours, was normal except on two occasions when it reached 99.6 orally. Surgical consultation considered the patient a poor risk for any surgical intervention because of his age and the extent of his pulmonary disease.

At the patients' insistence to return home, he was discharged from the hospital on January 27, and maintained on the same therapy. He was readmitted on February 1, because of his inability to take care of himself at home. Dyspnea and weakness again became severe. Fluid levels remained unchanged on radiologic study of the chest. Medical management was reinstituted but a gradual deterioration in his condition was observed. During the 48 hours prior to his death, hypoxia, with lethargy and cyanosis became apparent. A tracheostomy was performed to facilitate the removal of bronchial secretions. A thoracotomy tube was inserted at the right fifth intercostal space in an attempt to re-expand the compressed good lung tissue by negative pressure suction. The bullous space was entered during this procedure and 200 cc. of grayish watery, non purulent fluid was aspirated.

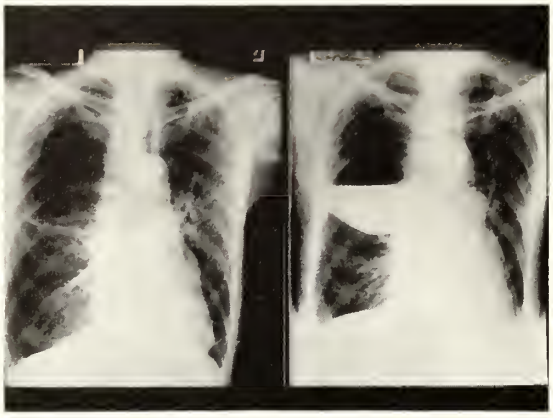


FIG. 1 FIG. 2

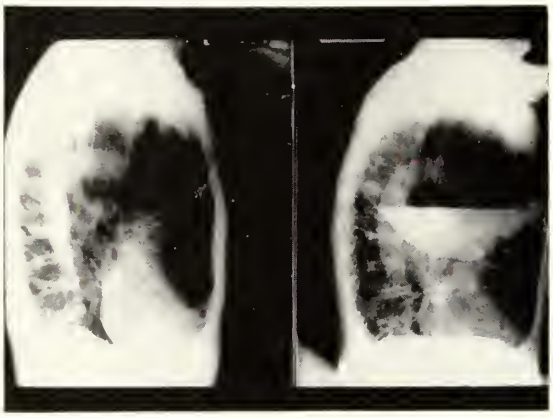


FIG. 1 FIG. 2

Fig. 1. Posteroanterior and lateral roentgenograms of the chest in June, 1962 shows a large bulla occupying the region of the right upper lobe and a few smaller bullae at the left apex. The heart and mediastinal structures are displaced across the midline to the left. There is compression atelectasis of the remainder of the right lung and the upper left lung. The diaphragms are flattened bilaterally.

Fig. 2. Examination of the chest in January, 1963 again shows the previously described bulla in the right upper lung field. At this time however, there was multiple air fluid levels in this region filling approximately one third of the cavity. There is increased thickening of the wall of the bulla. The remaining lung is unchanged from the previous examination.

In spite of these emergency attempts, the patients condition continued to deteriorate and he expired on February 9, 1963.

Autopsy Report: On gross examination, the upper right pleural cavity was obliterated by dense old fibrous adhesions. The previously noted drainage tube extended across the pleura into a large cyst in the right lung. The combined weight of the lungs was 1290 grams. The upper lobe of the right lung was virtually replaced by a huge, thick walled cyst that communicated with a bronchus and contained 350 cc. of greenish fluid. The middle and lower lobes of the right lung were compressed by this cyst but were not consolidated. The left lung had several smaller bullae scattered through all of its lobes, ranging in diameter from a few millimeters to two centimeters. The bronchi were patent throughout and showed no evidence of neoplasms. The pulmonary arteries were also patent and there were no infarcts.

On microscopic studies of the lung, there were areas of fibrosis, with infiltration of mononuclear cells throughout the parenchyma. Granulomas were noted consistent with tubercles and also a small cavity lined by caseous debris was found in the juxta bullous portion of the right lung. The wall of the large bulla consisted of dense, hyalinized collagen, with chronic inflammatory changes. Special stains of the lung tissue were positive for acid fast bacilli and negative for fungi.

Bacteriologic culture of the bullous fluid was negative for organisms.

Final Diagnosis:

1. Emphysema, bullous both lungs.
2. Cyst (bullous) upper lobe of right lung.
3. Tuberculosis, chronic fibro-ulcerative, lungs.

Principal cause of death: Respiratory failure secondary to chronic lung disease.

DISCUSSION

1. Definition, Pathogenesis and Pathophysiology of Emphysematous Bullae:

The emphysematous bulla is an air containing space within the lung substance which is lined by compressed alveolar walls or by a thin layer of fibrous tissue.[14] It is characterized by gross enlargement of pulmonary air spaces caused by distention and breakdown of bronchoalveolar substance and preservation of anatomic connections with the tracheo-bronchial air passages.[12] The bulla rarely contains fluid and in most instances has an indirect communication with the bronchial tree.[14,17]

The emphysematous bulla is distinguished pathologically from the congenital pulmonary cyst which is a fibrous walled, cuboidal or columnar epithelial lined cavity, having a fairly direct communication with the larger branches of the bronchial tree.[4,14]

As an acquired disease, emphysematous bullae may occur at any age but are more commonly found in the middle age male.[15] The mechanism for bullous formation is obscure but generally it has been attributed to a check valve phenomenon produced by inflammatory changes within the bronchiolar substance, and from loss of supporting elastic tissue within the alveoli, alveolar ducts, and respiratory bronchioles.[3,17,21] The force enlarging these bullae appears to be the elastic contraction of the normal lung pulling apart the flaccid degenerated tissue.[12]

The pathophysiologic effect of emphysematous bullae is the compression of good lung tissue and the utilization of a portion of the air exchange and the pulmonary blood flow without adding to the exchange of gases.[9]

Pneumothorax is a common complication of bullae but infection in bullous spaces is unusual.[18]

II. Development of infected Bullae:

Emphysematous bullae have been associated with pulmonary infections in the following manner:

A. They have developed during acute infections.[7] Influenzal and atypical viral pneumonias, as well as acute bacterial infections of the lung have all been implicated in producing bullous emphysema.[2]

B. They have resulted from the healing of tuberculous disease.[7] Pulmonary emphysema as a sequel and complication of all forms of pulmonary tuber-

culosis has been known for years. In the majority of instances these emphysematous changes are localized to the immediate vicinity of the tuberculous lesion. Emphysematous bullae are a common finding in tuberculous lungs, especially in those with fibrotic changes. These bullae usually remain unchanged in size and shape over long periods of time.[8] However, there are rare instances in which giant bullae with elevated intra-luminal pressures have been formed by the tuberculous process. These giant bullae may act as an artificial pneumothorax in producing closure of the adjacent tuberculous cavity.[11] Dobric,[5] in 1938, reported such a case in a 33-year-old male who several months after having an active tuberculous cavity was found to have disappearance of this cavity and its replacement by giant bullous emphysema. Leder and Zahn[11] in 1950, also reported a case of a 56-year-old male who over a ten-year period had gradual compression and disappearance of a tuberculous cavity by a giant bulla. In recent years, bullous formation has been described following the treatment of tuberculosis with antibiotic and chemotherapy.[1] The mechanism of bullous formation in this case was proposed by Alarcon[1] to be due to the cleansing effect of the chemotherapeutic agents with expulsion of caseous material. The evacuated cavity then expands by the normal thoracic traction acting on the damaged alveolar walls in the affected areas and by the fixation of the lung surface to the chest wall preventing an adequate elastic recoil of the lung.

C. Established emphysematous bullae have become secondarily infected by pyogenic organisms or tuberculs bacilli.[7]

1. Pyogenic infection as a cause for accumulation of fluid in preexisting emphysematous bullae is an uncommon finding, only a few reports in the world literature were noted by Bersack.[2] In the past decade in this country there were five cases by Bersack,[2] (1960), one case by Weisel,[20] (1950), and two cases by Rothstein,[14] (1954). In addition, four cases were reported from England by Golberg,[7] (1959). In general, the clinical features of the pyogenic bullae are attributed to the adjacent pneumonic process itself.[7] The patient usually shows signs and symptoms of an acute respiratory infection, although chronically infected bullae may be an unsuspected finding, as demonstrated in Rothstein's[14] two cases. The newly formed air fluid cavities generally disappear spontaneously after the subsidence of the pneumonia.[2] Usually, only some linear fibrotic strands remain to mark the site of infection. However, large preexisting bullae may persist even though the fluid disappears.[2] The X-ray appearance of pyogenic bullae may suggest multiple cavitation or abscess formation.[7] In fact, during the stage when the surrounding lung is consolidated it may be impossible to rule out an abscess.[2] Bersack[2] lists the roentgenologic features in favor of bullae as:
   A. Rapid appearance and extension of an air cavity.
   B. Sharp inner margin in the earliest phase.
   C. Fairly rapid changes of the fluid collection without expectoration of putrid material.
   D. Relatively slight involvement of the lung immediately surrounding the air cavity.
   E. A rather low fluid level – the presence of a high fluid level is more in favor of suppuration.
2. The formation of air fluid levels in preexisting emphysematous bullae secondary to tuberculosis of the adjacent lung tissue is a rare phenomenon. Rothstein[14] reported eight cases in 1954, and was the first to point out the peculiar behavior of a bulla containing area which becomes secondarily tuberculous.[13] All eight of his cases were in middle aged males who had a history of "asthma" or "chronic bronchitis" and also had contracted pulmonary tuberculosis at an earlier time in their lives. In these cases, the development of inflammatory changes and fluid levels in the bullae was asymptomatic. Fever, chills and increased expectoration were not observed. The chief pathologic features were the extension of infection along the interbullous septa, the development of fluid levels within the bullae and the gradual obliteration of the bullae with absorption of their contents. However, when tuberculosis first developed in the juxta bullous portion of the lung it had no distinguishing characteristics. Once the walls of the bullae were involved, the radiologic picture was comparable to that of pyogenically infected bullae. The bullae themselves remained radiolucent but the adjacent infiltration within the interbullous septa or into the surrounding lung tissue simulated the picture of multiple extensive cavitation or abscess formation. A knowledge of preexisting bullae in the involved lung area served to differentiate the infected bulla from these later conditions.[13,14] According to Rothstein,[13] the development of tuberculosis in preexisting bullae does not indicate an unfavorable prognosis but rather shows the activity of the tuberculous process. The recently reported case by Sanford[16] of a 35-year-old male with combined bullous emphysema and active pulmonary tuberculosis was in agreement with Rothstein's[13,14] observations in that no untoward effect resulted from fluid collection within the bulla. However, Golberg,[6] in 1956, reported a case of a 71-year-old male,

with advanced emphysema and a tuberculous bulla, who expired approximately two weeks after the onset of his illness. In contrast to the previous reports, this case showed that a superimposed tuberculous infection upon advanced emphysema with diminished pulmonary reserve, had led to pulmonary insufficiency and death.

III. Comments:

The difficulties in diagnosing tuberculosis in the elderly patient has been discussed elsewhere.[10,19] The main problems have been enumerated as follows:

1. Severe tuberculosis may lead to death of the patient before the investigation is completed.
2. Tuberculosis bronchopneumonia may be overlooked if acid fast bacilli are not found in a routine sputum examination and some other pathogen is recovered.
3. Localizing symptoms and signs of tuberculosis may be lacking in the very old.
4. In the presence of some other disease concomitant tuberculosis may go unnoticed.[19]

The present case report exemplified many of these problems. A chronically ill, elderly patient with advanced bullous emphysema developed a superimposed respiratory infection resulting in pulmonary insufficiency and finally death. A history of previous tuberculosis was not obtained. Clinically, fever and a productive cough were minimal and there was an absence of night sweats and hemoptysis. The initial diagnostic study was of little help in finding the etiology of the patients infection. The tuberculin skin test, sputum for acid fast and bacterial organisms as well as blood cultures were negative. The radiologic study showed an air fluid level within a preexisting bulla as the only significant change from previous X-ray reports.

In spite of the lack of information, tuberculosis should have been more strongly suspected if we consider the previous observations that air-fluid levels in tuberculous bullae were generally asymptomatic and that certain tuberculous lesions might not be observed radiologically. On the other hand, pyogenic bullae were more apt to reveal acute changes both clinically and radiologically. The development of air-fluid levels in this case probably represented a valid sign of tuberculous activity. If more significance were given to this one sign, then a more thorough search for acid fast organisms would have followed. Institutions of specific therapy might not have saved this patient's life but in any event exposure to tubercle bacilli would have been kept to a minimum by adequate isolation technique.

Summary

1. The undiagnosed tuberculosis patient in the open hospital ward presents a real threat to the health of all concerned.
2. A recent case of undiagnosed pulmonary tuberculosis associated with preexisting emphysematous bullae was described in which air-fluid levels within the bulla was the only indication of tuberculous activity. The patients clinical course and postmortem findings were presented.
3. Bullous emphysema was defined.
4. The literature on "infected bullae" was reviewed.
5. Comments were made concerning the difficulty in diagnosing tuberculosis in the elderly, and also the appearance of air-fluid levels in preexisting emphysematous bullae as a possible indication of pulmonary tuberculosis.

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144 State Street, Portland, Maine

The Role of the Diploma School of Nursing — 1966*

SISTER MARY CONSUELA**

It is a privilege to have been invited to participate in a review of the types of professional nursing education programs available in Portland today and to present some thoughts on how the nursing needs of our community and state are being and may be met in the immediate future. My focus will be on the part played by the Mercy Hospital School of Nursing. Cognizance of the disciplined mind of the physician and respect for your interest in getting to the heart of the matter has prompted me to eliminate the details of our educational program. I plan to focus briefly on several changes in society, which are affecting health care and especially nursing service, and therefore the objectives and goals of nursing education.

Four areas have been identified by a Committee on Perspectives appointed by the "National League of Nursing" three years ago.

These four areas are:

1. The nature of the patient himself; the composition of the patient population.
2. The corps of personnel involved in patient care, its composition roles, interrelationships, and its expertness.
3. The agencies through which nursing and health care are made available to the people.
4. The education of personnel who man the health services.

We have taken these into consideration when planning for the sequence of learning experiences in which our students are involved. First, I believe that it would be meaningful to review the admission policies which govern our careful selection of nursing students. All applicants have graduated from the college course or scientific course of an accredited high school and are in the upper third or half of the class. They have demonstrated strong average or above average academic ability, have supplied five character references including one from a teacher one from his principal or guidance director and one from a clergyman. Our applicants are personally interviewed and given an opportunity to discuss their reasons for selecting the three-year nursing program rather than the 2 or 4-year program, and for selecting the Mercy Hospital School of Nursing in particular.

Our program is the traditional 3-year one. There is a 5-week vacation period during each of the 3 years which makes it actually a 32-month program. The first year begins with concentrated classes on formal theory. A brief introduction to patients and the hospital environment starts in the 3rd week, and then gradually increases. By the beginning of the second year, the students have had carefully directed theory and practice of nursing care of patients with medical or surgical problems of a less complicated nature. The ratio of formal class to clinical experience moves from 2 to 4 to 8 to 32 hours. During this first year, we are concerned with assisting the student to become aware of herself and of her potentialities in nursing, to develop skill in working with patients as "people in trouble", to acquire beginning skills in administration of physical care and emotional support, and in the development of judgment based on sound scientific thinking.

During the 2nd year the program is in three sections: One 13-week period in psychiatric nursing at the Seton Institute in Baltimore, Maryland. One in a concentrated surgical nursing experience during which time teaching and directed experiences are obtained in the operating room, recovery room, emergency room and in caring for one patient immediately prior to and following surgery for a period of 24-36 hours. The remaining weeks are designed to augment the student's learning in the care of patients who are being treated for an orthopedic condition, a neurological condition or skin condition. The ratio of theory to clinical experience continues at about 1 to 4. Emphasis during this time is also given to the role of the professional nurse as the team member, contributing with other discipline to the plan of care for each patient in light of his individual needs.

The final year is divided into two major blocks: one in Maternal and Child Nursing, one in Advanced Medical-Surgical Nursing. We believe that these two experiences can be much better assimilated with the backgrounds of all of the previous experiences including psychiatric nursing. During the Maternal and Child Nursing experience the learning opportunities are expanded by use of community resources such as Cardiac Clinics, Well Child Conferences and visits to various schools providing for handicapped children. The Medical-Surgical experience includes 4 weeks in the special care unit, and in the care of patients on a therapeutic diet regime. During this time, we not only introduce new learning experiences but hope to assist the student in acquiring depth as well as breadth of nursing knowledge, in developing skills in identifying and solving nursing problems, and in arriving at sound judgments. The registered nurse of today works with many disciplines, each distinct but in many instances with overlapping of responsibilities. We believe our graduates need to have a beginning awareness of the broad health needs of society today in the world, in the nation and in the immediate locality. They should be cognizant of the role they play as professional nurses in planning that these needs be met. There are legal, moral, and professional responsibilities, all of which must be

Continued on Page 97

*Presented at November meeting of Medical Staff.

**Director of Nursing, Mercy Hospital, Portland, Maine.

Maine Medical Association

Program-in-Brief — 113th Annual Session

The Samoset – Rockland, Maine

Sunday – Monday – Tuesday

June 12, 13, 14, 1966

Sunday, June 12

9:30 A.M. First Meeting of the House of Delegates
Presentation of A. H. Robins' Physician Award for Community Service

Presentation of Parke, Davis & Company Historical paintings to John F. Dougherty, M.D., President, Maine Medical Association

12:30 P.M. Luncheon

3:00 P.M. Second Meeting of the House of Delegates

6:30 P.M. Dinner
Speaker: To be announced

Monday, June 13

9:30 to 12:00 NOON

9:30 A.M. **Diseases of the Esophagus**
JULIUS WOLF, M.D., Chief, Medical Service, Veterans Administration Hospital, Bronx, New York

10:30 A.M. **Panel – Management of Biliary Tract Disease and Associated Conditions**

Moderator: GEORGE J. ROBERTSON, M.D., Medical Director, Bingham Associates Fund and Senior Physician in Internal Medicine, Tufts-New England Medical Center, Boston, Massachusetts

Panel:

PERRY J. CULVER, M.D., Associate Physician, Massachusetts General Hospital, Boston, Massachusetts

KENNETH W. WARREN, M.D., Department of General Surgery, Lahey Clinic Foundation, Boston, Massachusetts

ROBERT E. WISE, M.D., Chairman, Department of Diagnostic Radiology, Lahey Clinic Foundation, Boston, Massachusetts

12:00 NOON to 2:00 P.M. Luncheon

1:30 to 4:00 P.M.
Scientific Program Sponsored by the Maine Chapter, American College of Surgeons and Maine Trauma Committee

1:30 P.M. Business Meeting, Maine Chapter American College of Surgeons

2:00 P.M. **Pain, Psychosomatics, and the Pancreas**
GEORGE L. NARDI, M.D., Visiting Surgeon, Massachusetts General Hospital and Associate Clinical Professor of Surgery, Harvard Medical School, Boston, Massachusetts

3:00 P.M. **Prevention and Treatment of Low Back Pain**
HANS KRAUS, M.D., Associate Professor of Physical Medicine, New York University School of Medicine, New York, New York

4:00 P.M. **General Assembly**
Election of President-elect

7:00 P.M. Annual Banquet
Speaker: HOMER H. STRYKER, M.D., President, Stryker Corporation, Kalamazoo, Michigan
Subject: **Backing Into Business Without Even Trying**

Presentation of Honorary Pins

Tuesday, June 14

9:30 A.M. to 12:00 NOON

9:30 A.M. **New Concepts in the Diagnosis and Prevention of Diabetes**
LEO P. KRALL, M.D., Senior Staff, Joslin Clinic and New England Deaconess Hospital, Boston, Massachusetts

10:30 A.M. **Panel – Medical Emergencies**
Moderator: GEORGE W. WOOD, III, M.D., Internist, Brewer

Panel:

EDWARD B. BABCOCK, M.D., Internist, Bangor

LLOYD BROWN, M.D., Surgeon, Bangor

HAROLD D. CROSS, M.D., General Practitioner, Hampden Highlands

WARREN G. STROUT, M.D., Anesthesiologist, Bangor

12:00 NOON to 2:00 P.M. Luncheon

2:00 to 4:00 P.M.
Program Sponsored by the Maine Medico-Legal Society
Program to be announced

6:30 P.M. Lobster Bake

SPECIALTY GROUP MEETINGS

Monday, June 13

2:00 to 4:00 P.M.

Section on Ophthalmology of the M.M.A.

Clinical Fundus Pathology

DAVID D. DONALDSON, M.D., Boston, Massachusetts

Maine Society of Clinical Hypnosis

Practical Applications of Hypnosis in General Medicine

HERBERT SPIEGEL, M.D., Assistant Clinical Professor of Psychiatry, College of Physicians and Surgeons, Columbia University, New York, New York

Maine Thoracic Society, Maine Society of Internal Medicine and Maine Society of Anesthesiology

Pre and Postoperative Care of Patients With Chronic Lung Disease

LEROY D. VANDAM, M.D., Clinical Professor of Anesthesia, Harvard Medical School, Boston, Massachusetts

Utilization, How Useful?

FRANK P. FOSTER, M.D., Department of Internal Medicine, Lahey Clinic Foundation, Boston, Massachusetts

Tuesday, June 14

10:00 A.M. Maine Medico-Legal Society

Business Meeting

12:00 NOON Maine Radiological Society

Luncheon Meeting

CHARLES W. CAPRON, M.D., Portland, presiding

Speaker: EDWARD B. D. NEUHAUSER, M.D., Radiologist-in-Chief, Children's Medical Center, Boston, Massachusetts

2:00 to 4:00 P.M.

Ear, Nose and Throat Group

Maine Society of Obstetrics and Gynecology

Business Meeting

FREDERICK B. LIDSTONE, M.D., Auburn, presiding

SPECIAL NOTICES

Sponsors

The speakers for the scientific programs are supported in part by grants from Merck Sharp & Dohme Postgraduate Program, Eli Lilly and Company State Medical Convention Program, Warner-Chilcott Laboratories and the Diabetes Detection Program of the State of Maine Department of Health and Welfare in cooperation with the Technical Exhibitors.

Mead Johnson Laboratories Aesculapius Award for Outstanding Scientific Exhibit

With the aim of supporting and encouraging the development of scientific exhibits of high quality, Mead Johnson Laboratories has created the Aesculapius Award, designed to recognize and reward excellence in concept, originality and execution of scientific exhibit presentations at the Annual Session of the M.M.A.

Under the terms of this program, the most outstanding scientific exhibit will be selected by a committee of the medical society. The author of the winning exhibit will be presented with an appropriate certificate and a cash prize of $200.

Election of President-elect

The election of a President-elect will take place at the General Assembly, June 13 at 4:00 p.m.

House of Delegates

Sunday, June 12 – 9:30 a.m. and 3:00 p.m.

Included in the Order of Business for the meetings of the House of Delegates will be final action on the Budget for 1967, Standing and Special Committee Reports, and the Nominating Committee Report.

Election of Councilors

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 12 at 3:00 p.m.

First District – Cumberland and York

Second District – Androscoggin, Franklin and Oxford

Golf Tournament

Daniel R. Shields, M.D., Lewiston, Chairman

Flagyl destroys trichomonads



here & here & here & here

No topical treatment can

Flagyl®
brand of
metronidazole

Flagyl eliminates the difficulties and frustrations that have long attended the treatment of trichomonal infection.

These difficulties arose mainly from:

1) the failure of any previously known agent to destroy the protozoan in paravaginal crypts and glands;

2) the failure of any previously known agent to prevent reinfection by eradicating the disease in male consorts.

The introduction of Flagyl removed both of these long-standing deficiencies. Hundreds of published investigations in thousands of patients have confirmed the ability of Flagyl to cure trichomoniasis.

Correctly used, with due attention to repeat courses of treatment for resistant, deep-seated invasion and to the presumption of reinfection from male consorts, Flagyl has repeatedly produced a cure rate of up to 100 per cent in large series of patients.

Nothing cures trichomoniasis like Flagyl.

Dosage and Administration

In *women:* one 250-mg. oral tablet t.i.d. for ten days. A vaginal insert of 500 mg. is available for local therapy when desired. When the inserts are used one vaginal insert should be placed high in the vaginal vault each day for ten days, and concurrently two oral tablets should be taken daily.

In *men:* in whom trichomonads have been demonstrated, one 250-mg. oral tablet b.i.d. for ten days.

Contraindications

Pregnancy; disease of the central nervous system; evidence or history of blood dyscrasia.

Precautions and Side Effects

Complete blood cell counts should be made before and after therapy, especially if a second course is necessary.

Infrequent and minor side effects include: nausea, unpleasant taste, furry tongue, headache, darkened urine, diarrhea, dizziness, dryness of mouth or vagina, skin rash, dysuria, depression, insomnia, edema. Elimination of trichomonads may aggravate moniliasis.

Dosage Forms

Oral–250-mg.tablets/Vaginal–500-mg.inserts

SEARLE

Research in the Service of Medicine

Special Article

The Warning Label and the Physician

Mr. George T. Nilson*

By federal legislative act, beginning January 1, 1966, all cigarette packages must bear the label: "Caution: Cigarette Smoking May be Hazardous to your Health." Without attempting to assess the merits of this action by the government, which also prohibits any extension of this warning to cigarette advertising until 1969, we believe it is apparent that the physician's role in safeguarding the public health from the ill effects of cigarette smoking has become more urgent than ever.

There can be little doubt but that further action by federal and state legislatures will eventually be taken to reduce death rates due to the effects of inhaled tobacco smoke. What form this action will take depends upon several other factors — not the least of which is the degree of responsibility for the nation's health assumed by the medical profession.

Despite an enormous propaganda effort by the tobacco industry, the facts about the health hazards of cigarette smoking have been widely accepted by the medical and other health professions. Thousands fewer physicians are smoking cigarettes today than a few years ago, and many are participating actively with voluntary and public health organizations in helping cigarette smokers to rid themselves of the habit. These are valuable assets in reducing the death toll from disease associated with cigarette smoking, but the fact remains that the physician looms as the most important individual figure in improving the nation's health.

Surely if upwards of 300,000 lives are lost annually because cigarette smokers are helplessly trapped in the tangle of the cigarette habit (or addiction), the physician must reckon with this thought every time he treats a patient who will not, or cannot, rid himself of this threat to his life.

This is the personal responsibility of every physician in America. If he cannot meet this challenge now, it seems that government — perhaps slowly, less effectively, but most surely — will move to protect the smoking public from its own follies.

What is needed is a prompt action by the medical profession, but most of all, individual and decisive action by the practicing physician in his own community and his own practice.

* Field Director, Bingham Associates Fund, Augusta, Maine.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Public Health in Transition — Part I*

HARRY ALLEN, M.D.**

FOREWORD

The far reaching effects of new Federal health legislation and the major problems being encountered in its implementation are outlined so comprehensively in this paper by Dr. Allen that the information should be of interest and value to the medical profession. Dr. Allen and his staff are working closely with State of Maine Health and Welfare officials in setting up the state agency's role in the major new programs — Dean Fisher, M.D., Commissioner.

It was with considerable misgiving that I accepted the invitation to discuss some of the implications of the 1965 Legislative landslide in the health field. Since most of the programs are in the developmental stage, one is in the position of making supposedly intelligent predictions, with full knowledge that yet-to-be-released regulations and standards may completely invalidate the statements. In spite of this insecurity, it is extremely exciting to be a part of the dynamic changes we are experiencing in public health.

The Social Security Amendments are of major interest and have been predicted to have an impact on total medical care equivalent to the discovery of penicillin. However, careful evaluation and review indicates that Medicare is only one part of a series of fundamental changes occurring in concept, administration and delivery of comprehensive health services in this country.

It has become apparent that the traditional Federal-State-Local relations have not been adequate to meet the new dimensions in public health. There has been increasing dominance of the health scene at the Federal level with concurrent demand that the States assume new functions and responsibilities. This has placed the State and local Health Departments at the crossroads of their very existence as functioning, dynamic units in the health care of our citizens.

The following factors are contributory to this situation:

1. Congressional support of Health Legislation.
2. Inadequate legislative support at the State level.
3. The categorical grant system.
4. "Horizontal Programming" on the Federal level.

FEDERAL HEALTH DOLLAR

The budget for HEW is now second only to the Department of Defense. The total health program has had the continued enthusiastic support of Congress. More money is now available for professional education, research, environmental health, public health and related programs than at any other time in history. The new Legislation implies a national declaration to provide top quality health care in an optimum environment that is available to everyone, in every State and every neighborhood.

INADEQUATE STATE LEGISLATIVE SUPPORT

The national mandate has placed the States in a critical situation since the Federal demands have *not* been accompanied by increasing legislative appropriations to employ the necessary staff to develop new programs. The majority of State Legislatures appear reluctant to increase support for health programs. As a result, many of the States have not developed past the "Basic Six Functions of a Health Department" propounded by Dr. Haven Emerson in the late thirties. Some have steadfastly avoided the entire field of medical care and direct service-oriented programs.

CATEGORICAL GRANTS

Over the past several years, new Federal grants earmarked for Heart, Cancer, Chronic Illness and Aging, etc., have been made available. This categorical system had as a basis the experience with communicable diseases, and constitutes a system based on the past tradition of public health. It is obvious to all of us in this grant business that *a different approach is needed to effectively deal with the dominant health problems of today.* Comprehensive programming includes all services ranging from prevention, diagnosis, treatment and rehabilitation and cuts across several traditional operational programs in the usual State and local Health Department. An expanded base is essential for mounting effective programs, at both the Federal and State and local levels. A major result of the categorical grant program is costly fragmentation and duplication.

HORIZONTAL PROGRAMMING

It is becoming increasingly apparent that *Health programs are no longer the specific domain of a single established unit in the Bureaucratic structure.* Many new programs and grants relating to health are being initiated by other agencies, some even in competition

*Part I of a paper presented by Dr. Allen at the Harvard School of Public Health, Cambridge, Massachusetts, January 10, 1966. Part II will be published in the next issue of The Journal.

**Regional Program Director for Chronic Diseases, Public Health Service, U.S. Dept. of Health, Education and Welfare, Boston Regional Office, Region I, 120 Boylston St., Boston, Massachusetts.

with existing grant programs. Examples of these are Office of Economic Opportunity, Office of Aging, Department of Labor, Department of Education, Children's Bureau, Space Administration as well as the National Institutes of Health within the Public Health Service. These organizations have directed their efforts toward a multiplicity of agencies at both the State and local levels. This lack of coordination has resulted in some confusion, but fortunately not chaos. However, in terms of health benefits *"the sum is not equal to the parts."*

The problems posed by the above situation were brought squarely into focus by three recent pieces of Legislation:

1. Mental Retardation Planning.
2. President's Commission on Heart, Cancer and Stroke.
3. Medicare.

As you probably know, the Mental Retardation Legislation, passed in 1964, had the Kennedy Family's backing as the driving force. The most significant aspect of this program was the entirely new approach to community planning for health programs. This is now referred to as the "Umbrella Concept". Basic to the concept is:

1. Single agency designation by the Governor as the sole agency for carrying out the required program planning.
2. Involvement of all agencies, including the Health Department mental health agencies, medical schools, other public and private organizations, and representatives of the consumers in the planning process.
3. Development of comprehensive State and areawide health services, including manpower and facilities.

Mental Retardation Program planning posed a challenge for the Health Departments as well as the Federal agencies to work together. This was not accomplished without a degree of frustration by many persons accustomed to implementing programs through established channels. However, *the critical need for strengthening the planning process and developing coordinated, meaningful health-action systems was long overdue.*

The Mental Retardation program was but another indication that the *State Health Departments could no longer be considered the sole representatives for health program development in a given State,* and that comprehensive multi-agency planning was the new approach to meeting current and emerging health service needs.

The proposed Regional Medical Programs for Heart, Cancer, Stroke (and related diseases) also put the traditional public health administrators in the new role of working and planning with medical schools and private medical practitioners. Several States have widened their horizons and have seen the tremendous opportunity afforded in adapting to the current trends as indicated by the new health legislation.

Medicare provides the public health profession and the official agencies an opportunity to produce or to be phased into functional inactivity–to adapt to new situations and demands or stay at the Basic Six Level. *The State Health Agency is in the medical care business* and in a position to promote quality medical care for all the people. Money is available from the Federal Government to employ the adequate staff required to perform the legal responsibilities under the Legislation. Undoubtedly, no previous health program is more complex and yet more challenging to the Health Departments than the implementation of the Social Security Amendments of 1965.

It is not my intention to go into the details of the Medicare Bill which you can read at your leisure. In very brief outline, there are two major amendments to the Social Security Act, Title 18 and Title 19.

Title 18–The Health Insurance Program which we will consider at this point of our discussion consists of:

Part A – *Basic Hospital Plan*

1. Inpatient hospital services.
2. Post-hospital extended care.
3. Outpatient hospital diagnostic services.
4. Post-hospital home health services.
5. Inpatient psychiatric hospital services.

Part B – *Physicians Services*

1. Physician and Surgeon services at the hospital, clinic or home.
2. Home health services.
3. Diagnostic X-ray, laboratory.
4. X-ray, radium, etc., therapy.
5. Ambulance service.
6. Appliances.

The administrative details that are presently being worked out should be of some interest. In New England, as in the nation as a whole, the State Health Agency has been delegated administrative responsibility in connection with Title 18. (One exception is the combined designation of Health and Welfare in Rhode Island.) The Social Security Administration is working closely with the Public Health Service to secure the following two documents:

1. A Contract with State Health authorities to support the agency role in certification and consultation.
2. Home Health Services formula grant.

It should be noted that the Public Health Service received considerable amounts of money from Congress to assist and support the Health Departments in carrying out their legal obligations. To accomplish this objective, the funds appropriated included support for a new Division of Medical Care Administration.

The pressure of getting the program off the ground falls to the State Health Departments. A major part of the State's job includes:

1. Certification of hospitals, extended care facilities, home health services and laboratories.
2. Consultation.
3. Provision of direct service.
4. Supervision.
5. Function as intermediary.
6. Participation in Utilization Reviews.
7. Licensing.

The job of certification and consultation is by necessity a long-range program effort. However, the immediate goal is to attain maximum coverage by July, 1966. *The job will not even get off the ground unless the States acquire the necessary staff.* To assist the States, the Social Security Administration is contracting to underwrite the costs of all personnel needed to carry out this role. Currently, the States are recruiting personnel and approvable plans have already been submitted by five out of the six New England States. The approved plan provides *a major opportunity for the Health Departments to secure much needed staff* from the Social Security "Trust Fund." However, the shortage of skilled professional personnel and the inability to attract them

at the existing salaries offered by some State Merit Systems will create a major roadblock. This is true in three out of the six New England States (Maine, New Hampshire and Vermont.)

Another problem situation that remains to be solved in several States is the *lack of legal authority to collect fees and retain them for agency operations.* In some States all fees collected go into the general revenue. Likewise, the authority to certify and license laboratories needs clarifying in many States.

As previously mentioned, the States have an almost impossible deadline to meet. In the case of hospitals, the task is simplified by the fact that under the law, hospitals accredited by the Joint Commission on Accreditation of Hospitals are considered certifiable. A problem may face smaller hospitals in developing the required Utilization Review Procedure.

It is to be expected that many providers of service will feel somewhat threatened by the required Utilization Review Procedures. However, *an adequate review program will insure high quality patient care while making maximum use of existing resources.*

The implications of the hospital program are many and varied. The most significant will be the accelerated development of *comprehensive hospital-centered community care.* The term "Comprehensive Community Care" is employed to describe the interrelationship and utilization of medical care facilities. In this concept the community hospital becomes the hub of all care which radiates to and from it.

Increased utilization will encourage the hospitals to expand and improve outpatient diagnostic and treatment facilities. In addition, in many localities the hospitals will expand their present scope to include the provision of out-of-hospital home health service similar to the pioneering programs initiated in 1948 by the Montefiore Hospital in New York City.

It is also anticipated that many hospitals will develop extended care facilities to accommodate the chronically ill and aged. This may take the form of a separate facility or may be an integral part of the hospital specifically designed for long-term care. Thus, it is possible for a hospital to be certified as a provider of the three levels of service recognized by the Act.

The demand for professional staff including nurses, physical occupational and speech therapists, and medical social workers will also create problems for the hospitals by establishing a seller's market. This will result in the physician spending a greater portion of his time within the hospital. The trend toward a closer tie between doctors and hospitals is already a fact of life, but the process will be accelerated by Medicare.

The role of the intermediary should be mentioned since they will be in a key position in promotion of quality care. It is predicted that the computers will provide comprehensive data which will permit realistic evaluation and formulation of standards for the provision of medical care in a manner previously unattainable. A side effect will be seen in the automation of appropriate hospital services. Some hospitals, such as Kaiser Permanente in California, are already involved in this streamlining of outpatient and laboratory facilities.

THE ROLE OF THE DIPLOMA SCHOOL OF NURSING – 1966 – *Continued from Page 89*

learned objectively and with opportunity to demonstrate their learning. Opportunity for developing leadership in planning patient care is afforded the student in the last months in the school. Many of our graduates return to smaller hospitals throughout the state and have limited immediate resources available to them for direction.

We recognize that the young high school graduate of today is looking for education leading to an academic degree. We recognize that many are eager to select a field of endeavor where they can find personal satisfaction and fulfillment by becoming involved in meeting health needs of people here and abroad, now and in the future. We endorse the philosophy which states that professional nursing rightfully belongs in institutions of higher learning; but we at Mercy, as in many of the 700 to 800 hospital diploma programs around the nation, are firmly convinced that the graduates of the diploma programs are essential in increased numbers during the next decade until some balance between the supply and demand for registered nurses can be established. You ask what we are doing to help meet the needs. Our answer is: 1) recruiting for nursing in all levels of education, advising the student in the light of her own interests and potential; 2) endeavoring to structure our curriculum on sound educational principles so as to teach for the future as well as the present and attract qualified persons; 3) participating in accreditation programs so as to be eligible for federal funds to assist the student financially; 4) investigating every opportunity available to expand our program for increased numbers that can be taught good patient care.

Before closing, I wish to acknowledge with sincere and deep appreciation the support and personal contribution the Medical Staff and the individual members have made on many occasions to the school of nursing. Your willingness to lecture periodically, to teach on an impromptu basis, your support of student projects and your recognition of the contribution of a student or young graduate are more influential than you realize. Your efforts to strengthen the practice of medicine, through research and a medical staff educational program here at Mercy, have a very immediate effect on the quality of patient care of which nursing is a significant part. Nursing students, like all students, learn more surely from what they see in practice than from what they learn or read in books. Therefore, my word of respect and appreciation, to you, for helping us to meet our goal of contributing to the provision of quality nursing care where ever our graduates practice, in Portland, in Maine, in the USA or abroad; in hospitals, nursing homes, armed forces, or in any spot where nurses are needed.

Maine Heart Association Notes

116 State Street, Augusta, Maine

Analysis of Experience in Workmen's Compensation for Heart Cases

Much concern, discussion, and misunderstanding about this situation is shared by physicians, insurance personnel, workmen, and employers. . . .

Most physicians find it difficult to treat a patient and assist in his recovery without becoming emotionally invested in him. . . . It is difficult for the claimant's physician to be an objective judge of the possible relation of an incident at work to a subsequent heart attack when medical knowledge about such relationship is inadequate, and when an adverse recommendation by the doctor may prevent the receipt of financial aid to which the patient and family believes they are entitled. . . . Indeed, if the patient is aware that a negative opinion by his doctor prevented such award, it may be impossible to continue the doctor-patient relationship. This undoubtedly accounts for the tendency of the claimant's physician to render an opinion suggesting causal relation. Thus, it appears that the attending physician, whether he is general practitioner, internist, or even cardiologist, should not be asked to make a judgment as to causal relationship on his own patient. This is an important reason for the existence of impartial panels. . . .

It is all too easy for the claimant, or even the physician, to associate two events closely related in time in a cause and effect relationship. . . . It is likely that just such a process is operative in many cardiac industrial cases. Cases still appear in which the physician states in essence, "The heart attack happened on the job, so the job must have caused it." (We never seem to see the obvious corollary, "The heart attack happened while he was in bed sleeping, therefore sleep must have caused it.") Such circumstantial reasoning is scientifically undesirable, is detrimental to the cost of maintaining an adequate insurance program for injuries clearly related to work; and it has a long-range effect of discouraging employers from hiring cardiac workers, or permitting cardiac patients to return to work. . . . Few if any will object to the acceptance of cardiac claims when the work may be clearly shown to have caused the cardiac episode. Furthermore, it is unlikely that employment opportunity will be damaged if awards are on a consistent basis which all may examine.

. . . criteria for guidance of physicians in the determination of causal relationship appear to be valuable, and determination of causal relationship and rating of disability may be better performed by a person other than the patient's attending physician.

Submitted by Jacob B. Dana, M.D.

Reference — Bergy, G. G. and Sparkman, D. R. Circulation, Volume 33, pages 461-473, 1966.

From the Secretary's Notebook

Summary of Proceedings, Interim Meeting, M.M.A. House of Delegates, April 3, 1966 at Brunswick, Maine

The interim meeting of the Maine Medical Association House of Delegates was called to order at 1:30 P.M. by the President-elect, George E. Sullivan, M.D. and presided over by Linus J. Stitham, M.D., Speaker of the House. There was a total attendance of 58, including 32 delegates, 12 alternates, 11 members of the Council, the Secretary-Treasurer and 2 guests.

A summary of this meeting has been sent to the President, Secretary and Delegates and Alternates of each County Medical Society and to the Officers and Councilors of the Maine Medical Association. Consequently, only a brief resumé will be published in the Journal.

The Financial Statement of Income and Expenditures for fiscal year ending December 31, 1965 and budget proposed for fiscal year ending December 31, 1967 was presented by the Budget Committee, Drs. Edward K. Morse and John D. Denison. The Budget Committee also proposed that the M.M.A. dues be increased in 1967 from $55.00 to $65.00 annually which would add approximately $7,700.00 to income from dues. *Final action on the budget proposed for 1967 and the proposed increase in dues will take place at the annual meeting of the House of Delegates on June 12 at The Samoset, Rockland, Maine.*

It was voted that the reports of the Ad Hoc Committee on Osteopathy go back to the county societies for discussion and that they be acted upon in June.

It was voted that we are not in favor of an increase in AMA dues and the delegate to AMA (Paul H. Pfeiffer, M.D.) was instructed to draw up a resolution to this effect for presentation at the annual meeting of the AMA House of Delegates in June.

A recommendation approved by Cumberland County Medical Society on March 24, 1966, which follows, was approved, i.e., it was voted that the President appoint the suggested committee.

> The Cumberland County Medical Society hereby recommends that a committee consisting of —— members of the Maine Medical Association House of Delegates be appointed by the President to investigate the feasibility of setting up a Special Membership in the M.M.A. for members who voluntarily retire at age 65 and that dues for this membership be prorated. We further recommend that proration of the State dues be determined by the M.M.A. House of Delegates and proration of county dues determined by the respective county societies.

A resolution re MEMPAC dues billing in conjunction with the "Society's annual dues billing statements" was rejected.

The President-elect announced the appointment of the following members to serve on the Nominating Committee: (First District) Howard P. Sawyer, Jr., M.D., Portland, (Second District) Albert P. Royal, Jr., M.D., Rumford, (Third District) Wesley N. Wasgatt, M.D., Rockland, (Fourth District) H. Carl Amrein, M.D., Madison – Chairman, (Fifth District) James C. Bates, M.D., Eastport, (Sixth District) Charles D. McEvoy, Jr., M.D., Bangor.

The report of the Nominating Committee shall be the first Order of Business at the Second Meeting of the House of Delegates on Sunday, June 12 at 3:00 P.M.

A brief report was presented by Charles A. Hannigan, M.D., Chairman of the Medical School for Maine Committee, relative to the feasibility study being made by Dr. John B. Truslow.

John T. Konecki, M.D. sent in a prepared report relative to Disaster Medical Care.

Stanley B. Sylvester, M.D., representing the Union Mutual Life Insurance Company stated that they plan a meeting with the M.M.A. Officers, Council and Health Insurance Committee on May 4, at which time they hope to have more information concerning Part B of the Medicare Program. (This meeting now scheduled for May 11.)

The Maine Cancer Society presented a check for $2,000.00 to the Maine Medical Education Foundation.

The members were reminded that election of Councilors for the following districts will take place at the Second Meeting of the House of Delegates on Sunday, June 12 – First District (Cumberland, York Counties), Second District (Androscoggin, Franklin, Oxford Counties).

The Executive Director, Dr. Hanley, called attention to Drug Abuse Control Amendments of 1965, establishing special federal controls for depressant and stimulating drugs which became effective February 1, 1966; increase in A.H.S. Group Insurance rates effective May 8, 1966; the Utilization Review for Maine Hospitals under Public Law 89-97 and recent developments concerning the Heart, Stroke and Cancer program.

The meeting adjourned at 4:00 P.M.

(Copy of the Summary of Proceedings of this meeting will be sent to any member of the Maine Medical Association on request.)

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Preoperative Irradiation in the Treatment of People With Cancer

Animal experiments have indicated that less-than-curative doses of x-rays given before surgery may increase the efficacy of operations for cancer.

It remains to be seen whether the results achieved with transplanted tumors in laboratory animals apply to human patients. But these findings in carefully controlled laboratory experiments lend significant support to the favorable reports that are emerging on the use of preoperative irradiation in the treatment of people with cancer.

This research under the direction of Dr. Bernard H. Feder of the Veterans Administration Hospital was reported recently by the American Cancer Society, which helps support it. Dr. Phyllis Blair, now of the University of California in Berkeley, collaborated in the work.

Four years ago Dr. Feder set out to determine whether x-rays would increase the beneficial effects of surgery in cancer.

Very often surgical removal of a cancer of the lung, breast, stomach, bowel or other organ seems successful, but months later cancer colonies crop up and kill the patient. Sometimes the cancers are so small they can not be detected during surgery: sometimes cancer cells, dislodged from the tumor during the operation, take up residence in the operative wound, or at a distant site, and grow into undetected deadly tumors (metastases).

Dr. Feder and Lynn Boutelle modified an ordinary shoemaker's needle into a transplanting instrument which works with almost 100 percent efficiency. The instrument can be plunged into a tumor, capture five to ten thousand cancer cells and readily transfer them to a normal animal's thigh. The entire procedure can be accomplished in less than thirty seconds, permitting the cancer cells to retain their potency.

With associates, Dr. Feder then began experimenting on two different kinds of transplantable mouse cancer. Untreated and spread with the needle, the cancers were almost 100 percent fatal.

Various doses of x-rays were tried. Large doses and small doses were tested; and the radiation exposure was administered in one large dose or a few or many small doses.

Numerous tests showed that the growth of transplantable mouse tumors is prevented by giving a moderate (not necessarily a cancer-killing) dose of x-rays to the tumor before transplantation is attempted.

The results suggest that in some instances light irradiation given prior to surgery in humans could prevent some of the recurrences of cancer.

American Cancer Society News Service Release

County Society Notes

PISCATAQUIS

The Piscataquis County Medical Society held a meeting at the Squaw Mountain Inn in Greenville, Maine on March 10, 1966.

George J. Robertson, M.D. of the Bingham Associates Fund was the guest speaker. He spoke on Rheumatic Fever and Rheumatic Arthritis. The talk brought all of us up to date on the etiology, diagnosis and treatment of the disease.

ISAAC NELSON, M.D., *Secretary*

LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on March 15, 1966. The meeting was called to order by the Secretary, George W. Bostwick, M.D.

Dr. Bostwick disclosed to the membership the reports from the Maine Medical Association announcing a prize for the best scientific exhibit at the annual session in Rockland. He requested the early submission of any resolutions which the society may wish to present at the Interim Meeting of the House of Delegates of the Maine Medical Association, and presented a resolution from the Maine Radiological Society supporting the policy of separate billing for professional fees by its members to patients to whom they supply professional services, and that charges for technical services connected with radiology should be separately billed by the hospital, institution, or group providing such technical services. This resolution was approved by the Council of the Maine Medical Association on February 13, 1966.

Fred S. Rosen, M.D. of Boston, Massachusetts was the guest speaker. He discussed serum proteins and the diseases connected with the abnormalities in their distribution.

GEORGE W. BOSTWICK, M.D., *Secretary*

CUMBERLAND

Seventy members and guests were present at the meeting of the Cumberland County Medical Society held at the Sheraton-Eastland Motor Hotel in Portland, Maine on March 17, 1966.

The President, Alvin A. Morrison, M.D., called to the attention of the membership the necessity of re-registering with the Board of Registration of Medicine prior to July 1, 1966. A letter was read from the United Community Services asking for reendorsement of the Information and Referral Center which had been given in 1964, and it was voted that the Executive Committee be authorized to act for the society in the matter after more investigation.

Dr. Morrison announced that George W. Hallett, Jr., M.D. had been appointed as the representative of the Cumberland County Medical Society to the Advisory Committee of Project Headstart. Dr. Morrison also indicated that he will appoint a committee to work with a similar committee from the Portland Dental Society to establish appropriate fees for services rendered by professional groups in federally financed projects of this type.

Francis A. Fagone, M.D. called attention to the VD Clinic which is being reactivated, and Dr. Morrison stated that Donald P. Cole, M.D. had agreed to be the society's advisor to the clinic.

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The society voted to accept the offer of Lederle Laboratories to sponsor a Lederle Symposium in Portland in 1967.

The availability of booklets dealing with office construction was announced, such booklets having been developed by The Sears-Roebuck Foundation and distributed by the American Medical Association.

The membership voted that Charles R. Glassmire, M.D. would be the nominee from the Cumberland County Medical Society to serve as the next District 1 member of the Council of the Maine Medical Association.

Merle S. Bacastow, M.D., appealed to the membership to learn all the possible facts about Medicare and the details of Utilization Committees, suggesting that it would be more advantageous to foresee the problems which will arise ahead of time than to have regulations prescribed by governmental agencies, not appreciative of local problems.

Dr. Morrison discussed the future problem regarding members of the Maine Medical Association who retire at age 65 and he read the proposal tentatively prepared for presentation at the April 3 meeting of the House of Delegates. The membership voted that such a proposal be made.

STANLEY B. SYLVESTER, M.D., *Secretary*

Announcements

Staff Activities – May 1966
Central Maine General Hospital
Lewiston, Maine

	May	
Friday	20	Medical Grand Rounds, 9:00 A.M.
Saturday	21	Medical-Surgical Grand Rounds, 8:30 A.M.
Wednesday	25	Journal Club, 11:00 A.M.
Thursday	26	Pediatric Grand Rounds, 9:00 A.M.
Friday	27	Medical Grand Rounds, 9:00 A.M.

Note: Unless otherwise indicated, meetings are held in the first floor Conference Room.

Workshop in the Comprehensive Application of Hypnosis in Therapy

A workshop in the Comprehensive Application of Hypnosis in Therapy, basic and advanced, will be held at the State University College at Oswego, New York, July 14-17. The workshop is directed by Lawrence M. Staples, D.M.D., who has directed many similar workshops in the past, and co-directed by George H. Estabrooks, Ph.D., visiting professor of psychology at the College at Oswego since retirement from the Colgate University Psychology Department. Frederick H. Ratzeburg, Ph.D., professor of psychology at the host college, will serve as coordinator. Following is the teaching staff: Leo Alexander, M.D., David B. Cheek, M.D., Donald Coulton, M.D., Milton H. Erickson, M.D., Lewis Jacobs, M.D., Frank A. Pattie, Ph.D., Bertha P. Rodger, M.D., Lawrence Staples, D.M.D. and Kay Thompson, D.D.S.

The sponsor is the American Society of Clinical Hypnosis. Registration for either the basic or advanced workshop is $75.00, with special rates for full time students, teachers, and interns. Registration is limited to dentists, physicians, and psychologists who are members of their national organizations. For more detailed information, write Dr. Frederick H. Ratzeburg, State University College, Oswego, N.Y. 13126.

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Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised; especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of pre-existing symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dose—operation of motor vehicles, machinery or other activity requiring alertness should be avoided. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia, the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor, and respiratory collapse.



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Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

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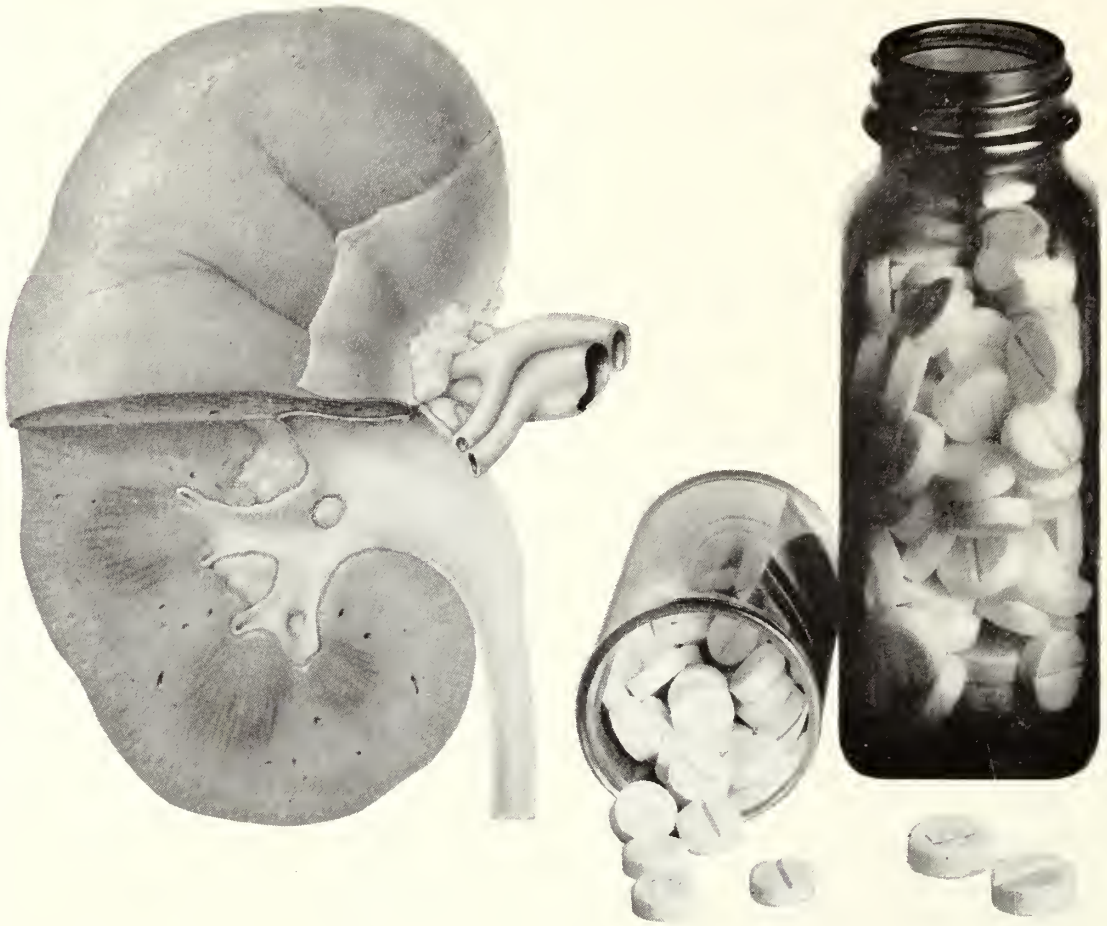
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For example, the Renal Research Program put fifteen years into this search before chlorothiazide became available. But because these years had first led to a greater understanding of basic problems, the desired criteria for chlorothiazide existed before the drug was developed.

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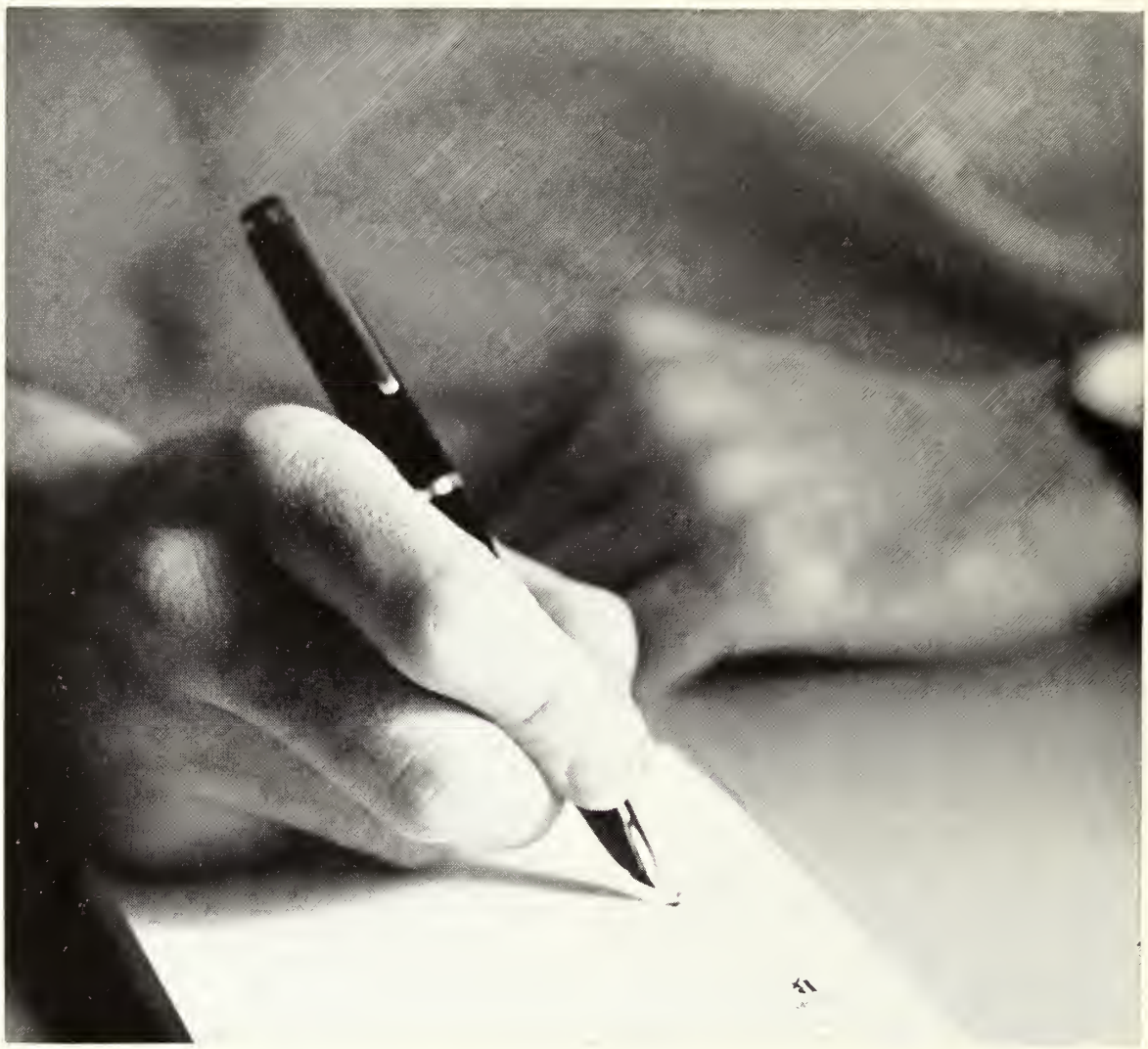
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Warning — In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of discomfort.

Precautions and Side Effects — Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncracy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg of demethylchlortetracycline HCl.

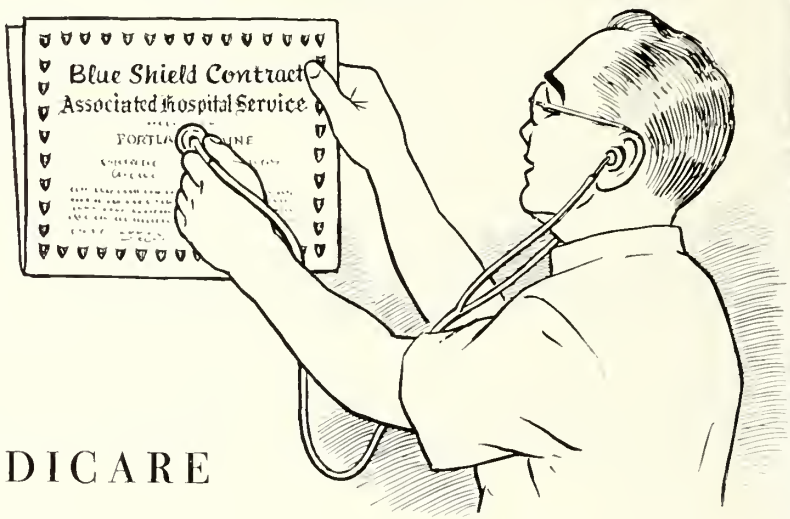
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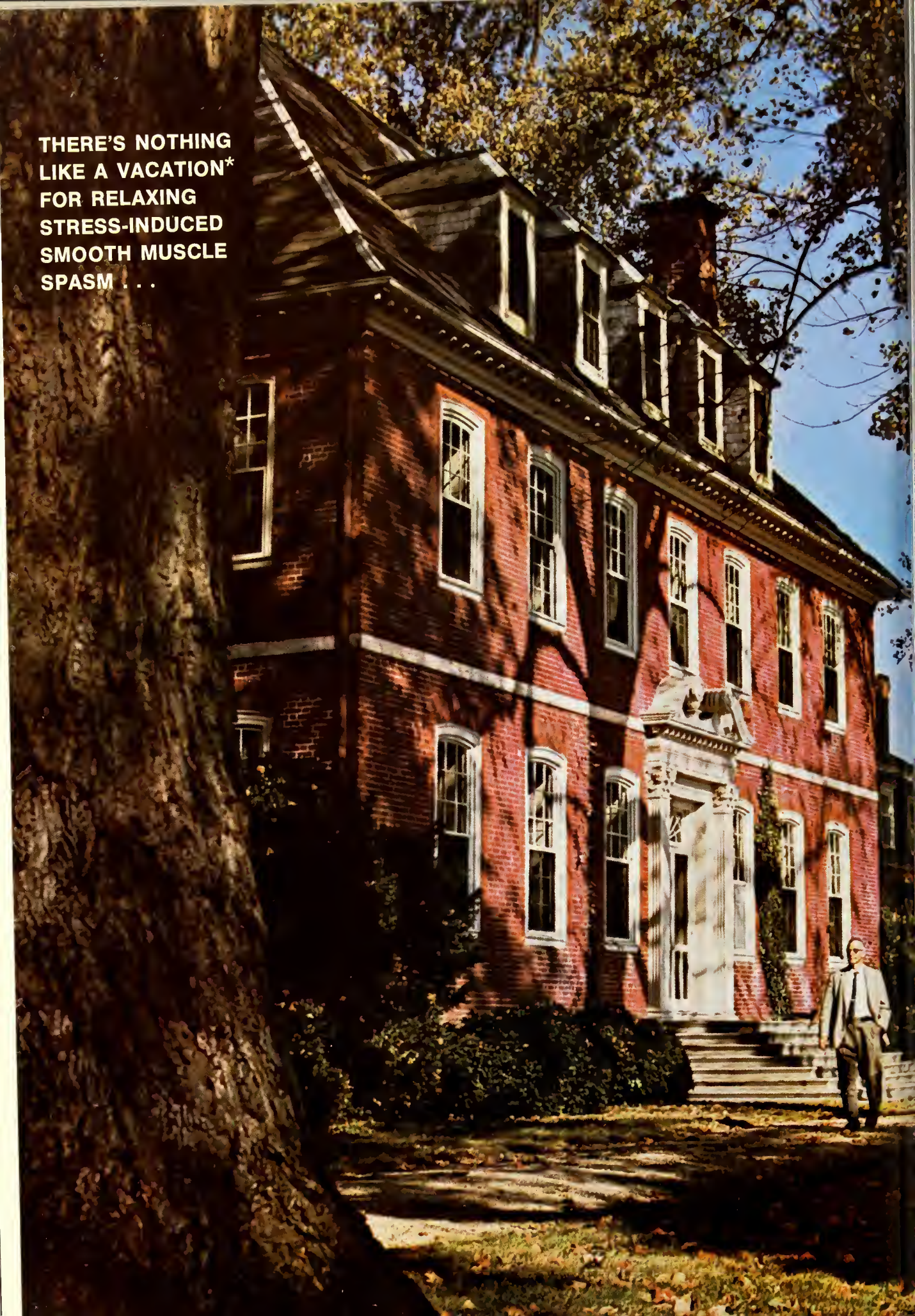
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Years of clinical use have established phenobarbital as one of the most efficient and highly regarded sedatives. In fact, for general sedation it is the drug of choice.[3] In Donnatal, phenobarbital potentiates the spasmolytic effects of the belladonna alkaloids, lessening emotional tensions and checking the neurogenic impulses that trigger GI disorders.

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Donnatal has withstood the test of time to become the classic sedative-antispasmodic because of its unsurpassed effectiveness, safety, economy, uniformity of composition, and dosage convenience. Its widespread acceptance and usage by the profession can also be attributed to its versatility in treating disorders characterized by smooth muscle spasm. There are more than two dozen distinct and separate indications for Donnatal listed in the current PDR.

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atropine sulfate	0.0194 mg.
hyoscine hydrobromide	0.0065 mg.
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(warning: may be habit forming)

IN EACH EXTENTAB

hyoscyamine sulfate	0.3111 mg.
atropine sulfate	0.0582 mg.
hyoscine hydrobromide	0.0195 mg.
phenobarbital	(3/4 gr.) 48.6 mg.

(warning: may be habit forming)

BRIEF SUMMARY: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Administer with caution to patients with incipient glaucoma, or urinary bladder neck obstruction. Contraindicated in acute glaucoma, advanced renal or hepatic disease, or a hypersensitivity to any of the ingredients.

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*This one at Westover, elegant Colonial Virginia plantation, located on the James River near Richmond. Built in the early 1730's by William Byrd II, founder of Richmond, it is now the home of Mrs. Bruce Crane Fisher.

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ial response is usually maintained dosage increases; indeed, initial is often reduced for maintenance es.

te or steroid therapy can usually be hed or, in some instances, eliminated.

ndications
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Before prescribing, the physician should obtain a detailed history and perform a complete physical and laboratory examination, including a blood count. The patient should be kept under close supervision and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Regular blood counts should be made. The drug should be used with greater care in the elderly.

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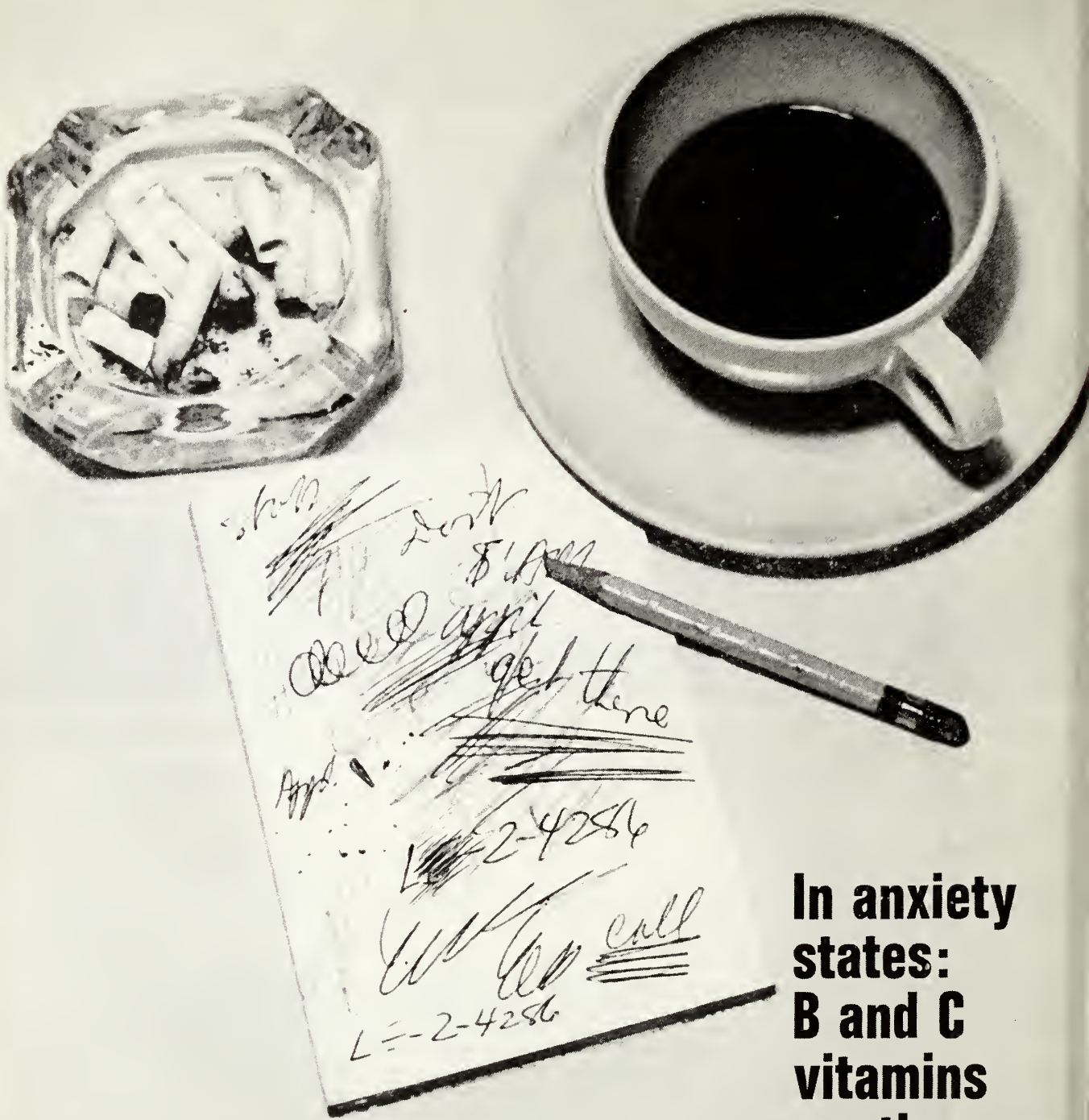
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Precautions: Advise patients against possibly hazardous procedures until maintenance dosage is established. Though compatible with most drugs, use care in combining with other psychotropics, particularly MAO inhibitors or phenothiazines; warn patients of possible combined effects with alcohol. Observe usual precautions in impaired renal or hepatic function, in long-term treatment and in presence of depression or suicidal tendencies. Exercise caution in administering drug to addiction-prone patients or those who might increase dosage; withdrawal symptoms, similar to those seen with barbiturates or meprobamate, can occur upon abrupt cessation after prolonged overdosage. Caution should be exercised in prescribing any therapeutic agent for pregnant patients.

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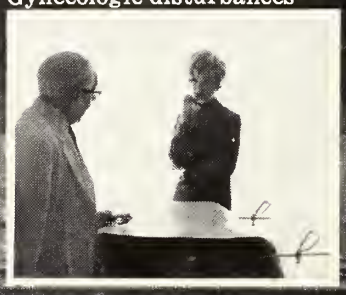
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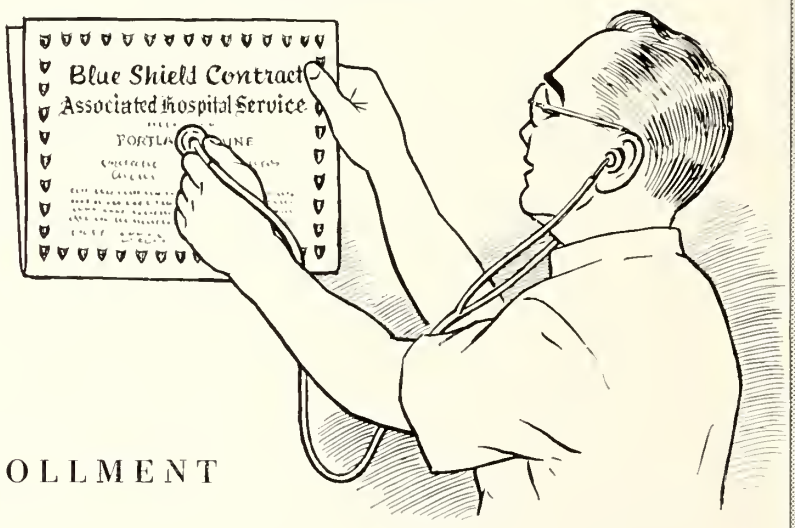
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this issue: partners in misery

Partners in misery: common cold and duodenal ulcer*



A century or two ago our forefathers believed that the common cold was caused by an excess of evil humors. Today we regard these baleful humors as a result of the cold rather than its cause. Only in the last ten years have some of the offending viruses been cultivated, and we now know many others can be transmitted to human volunteers although they cannot yet be grown in the laboratory. Various body defenses react to infection by a virus, and in the respiratory tract the lung is protected by the phagocytic properties of the pulmonary alveolar macrophage.[1]

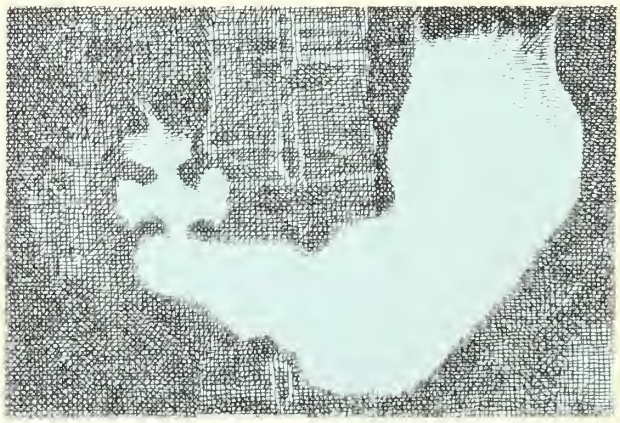
Because of the brief history and large proportion of unidentified viruses, effective prophylactic vaccines have not yet appeared. A universal antiviral agent seems even more remote. Meanwhile, these viruses continue to produce epidemics of upper respiratory infection at seasonal intervals, particularly in spring, fall and winter, during which they propagate and distribute progeny indiscriminately. As a result, they are responsible for significant work loss. Since the immunity afforded is temporary, recurrent infections are common.

This recurrent morbidity in spring and fall is shared by duodenal ulcer, itself a cause of significant loss in wages and medical expense. Thirty years ago Emery and Monroe reported 1,279 recurrences of peptic ulcer of which 13 per cent could be attributed to upper respiratory infection, thus confirming a long-held clinical impression.[2] While the inflammation, congestion and secretion of the mucosa of the nasopharynx in the common cold, and the hyperemia and hypersecretion of the gastric mucosa during acute exacerbation of a duodenal ulcer may not be directly linked, it is probable that the miseries of the infection are reflected in the pain of the ulcer. The following case history illustrates this point.

Case report A 48 year old white male executive, suffering from hematemesis and melena for 12 hours was admitted to hospital on April 8, 1965. He gave a history of duodenal ulcer proved by x-ray studies 23 years previously. In the intervening years he suffered from typical epigastric pain occurring two hours postprandially, usually for two or three weeks each spring and fall. A hospital admission two years earlier had followed hematemesis of moderate amount.

The present admission was preceded by a recurrence of pain during a period of unusual business pressure. In addition he caught a cold one week before his entry to hospital and his already irregular eating habits were made more so by loss of taste and appetite, blocked nose and general malaise. During the final 36 hours he used aspirin to relieve his cold, taking 2 or 3 tablets with a glass of water on 6 or 7 occasions. Before retiring he drank a glass of hot toddy.

He awakened about 1 a.m. and vomited dark red blood and recently ingested food mixed with whisky. Melena followed and he was transferred to hospital. On admission he was pale, cold and sweating. Apart from epigastric tenderness and black stool on the examining finger after rectal examination, his general physical examination was not remarkable. Temperature was normal, pulse was thin and thready with a rate of 114 beats per minute, and blood



The commonly found "clover-leaf" roentgenographic abnormality of chronic duodenal ulcer due to spastic contractions and scarring.



Typical site of duodenal ulcer showing anatomical relationship to gastroduodenal artery and common bile duct.

pressure measurement was 104/60mm. of mercury. Hemoglobin estimation was 8.6 grams per cent, but white blood count, chest x-ray and urinalysis were within normal limits. No further bleeding occurred. After blood transfusions and a suitable medical regime he was dismissed two weeks later and advised to return later for consideration of surgery.

The care of the ulcer patient suffering from a viral infection of the upper respiratory tract is additionally handicapped by the local and constitutional symptoms so produced. Indifference to food often makes it difficult to "feed a cold" even in a patient without an ulcer. In the ulcer patient this becomes more than a homily, for malaise and apathy towards irksome routine produce a lack of interest in adhering to an ulcer program and result in its eventual failure if these symptoms are not relieved. The absence of curative treatment for these viral infections has spawned many a strange therapeutic offspring and some of the more generally accepted remedies contain a special risk for the ulcer patient.

To the gastric physiologist, the shot heard around the world was not fired at Concord but was discharged through the stomach of Alexis St. Martin. The worthy voyageur had a more than modest thirst and on several occasions William Beaumont observed through the resulting fistula the changes of gastritis which followed an immoderate ingestion of alcohol.[3] It is now well known that alcohol stimulates the production of gastric juice and the patient with an ulcer, even in remission, should not drink alcohol without first taking food, milk or antacid. The use of various alcoholic remedies for the common cold is to be strongly discouraged in this type of patient. Factors already present can produce an acute exacerbation of ulcer symptoms without the added stimulus of alcohol.

Headache and facial pain, sore throat and generalized aching are subjective discomforts of viral infections of the upper respiratory tract or their complications. Relief from them is usually sought in the family medicine cabinet, and of all the medications habitually stored there, salicylates are the most frequently used. Oral ingestion of salicylates has produced



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substernal pyrosis and sometimes epigastric pain in susceptible individuals. Bleeding from erosions of the gastric mucosa has followed their use and has been severe enough in some instances to cause hematemesis and melena.[4] It has been suggested that interference with the protective mucous layer of the stomach allows this to happen. There is no evidence that hypersecretion occurs, but the taking of aspirin has been followed by exacerbation of ulcer symptoms and occasionally by gastrointestinal bleeding.[5] The ulcer patient with a cold should take salicylates with an antacid, or preferably other means of symptomatic relief should be found.

Caffeine has two actions on gastric secretion. One directly stimulates production of acid and the other potentiates the out-pouring of gastric juice as a response to other stimuli.[6] In high doses to animals, it has produced erosive gastritis and peptic ulceration. Caffeine-containing beverages are discouraged for the ulcer subject and forbidden during an acute exacerbation.[7] Many cold remedies contain caffeine.

To sufferers from allergic rhinitis springtime brings symptoms of nasal obstruction, loss of taste and smell and malaise similar to those caused by viral infection but due instead to allergens which at that time of year are principally tree pollens. Relief from these symptoms can be obtained, though not wisely, by the use of steroid medication. Unfortunately, chronic sufferers from these allergies have used this approach with varying degrees of success. Steroid hormones increase gastric secretion and delay the healing of experimental ulcers.[8,9] Clinically their administration has been associated with reactivation of healed duodenal ulcers, and with bleeding and perforation which were not always preceded by typical ulcer distress. Because of these harmful effects, their use in the ulcer patient is best avoided for other than serious medical problems and then only with adequate antacid coverage.

Summing up It is immaterial by which pathways the malaise and lassitude, depression and irritability activate an ulcer, but it is the physician's responsibility to ensure that the medications he selects to relieve the symptoms of a cold do not further aggravate the ulcer.

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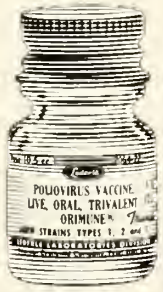
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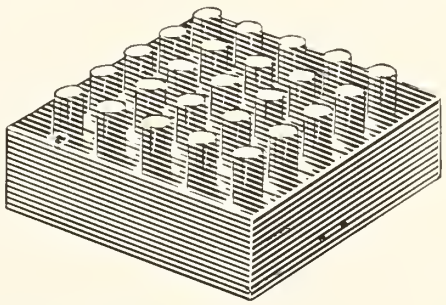


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*Rosenthal, S. R., Nikurs, L., Yordy, E., and Williams, W.: Scientific Exhibit Presented at the Annual Meeting of the National Tuberculosis Association, Chicago, Illinois, May 30-June 2, 1965.

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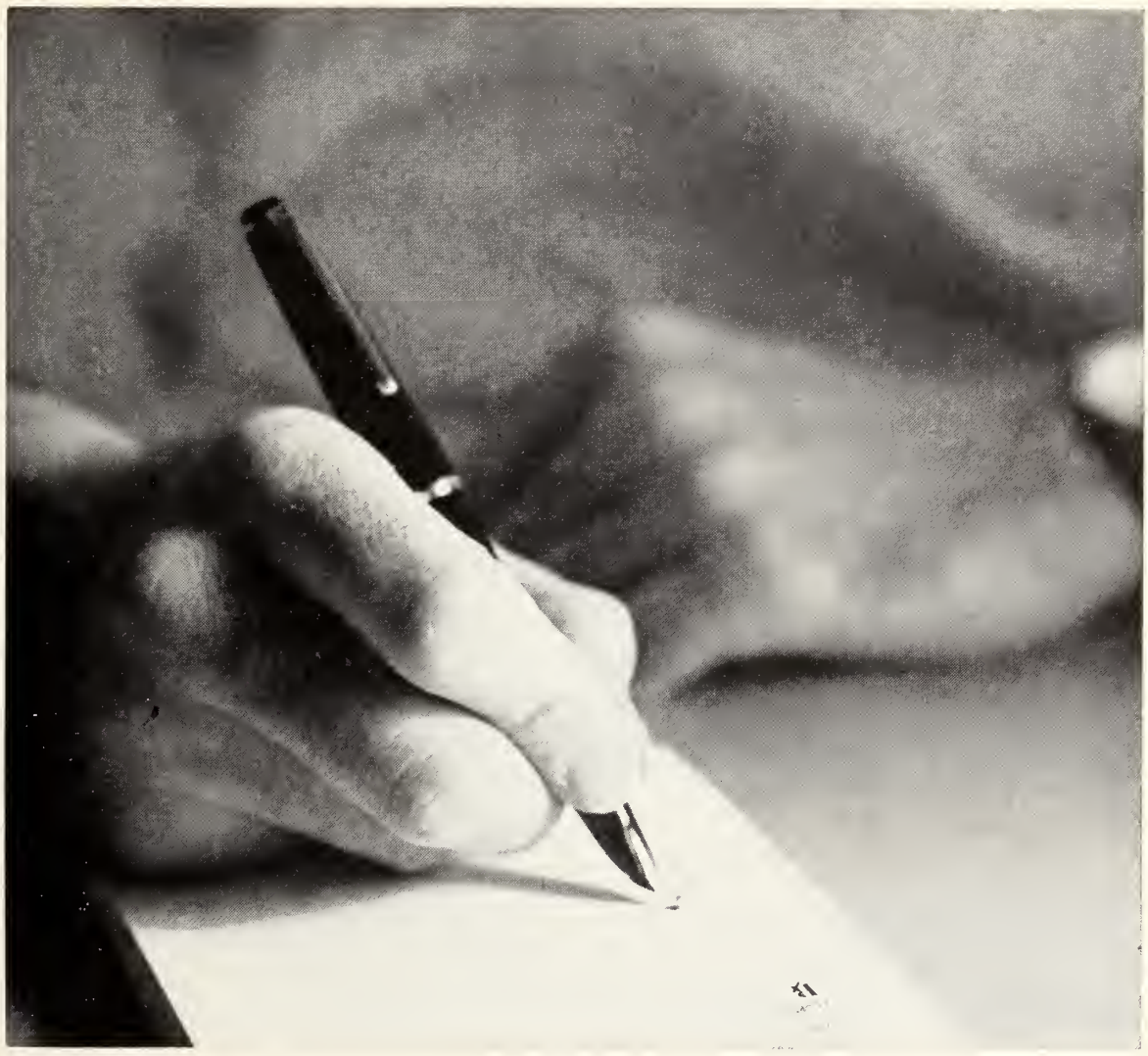
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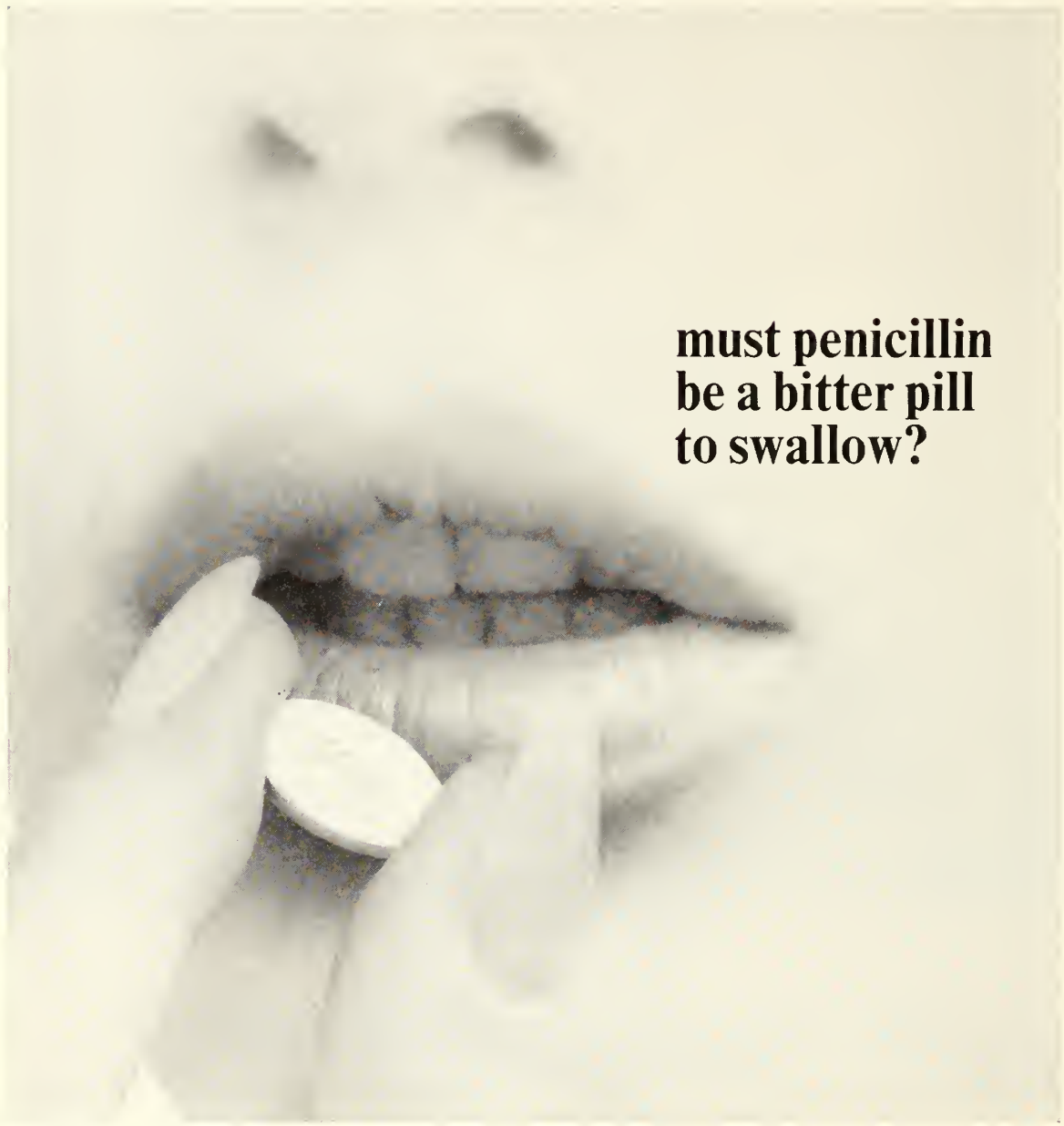
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Sur-Pak and Physical Radiation Protection Survey of Dental X-Ray Equipment in Maine† — Report of Findings — Project No. 932-2-6913

ALONZO H. GARCELON, D.D.S.* and JAMES W. MILLER, D.D.S.**

Coauthors: ELMER W. CAMPBELL, Dr.P.H., JAMES W. FULLER, M.P.H. and DONALD C. HOXIE, B.S.‡

The Dental Sur-Pak Radiation Protection Survey Plan was brought to the attention of the Maine Department of Health and Welfare in January of 1962. At that time Dr. E. W. Campbell, Director of the Division of Sanitary Engineering was attending a two-week course, entitled "The Medical Aspects of Radiological Health," and a Sur-Pak survey for the State of Maine was suggested to him by James W. Miller, D.D.S. of the State Assistance Branch, Division of Radiological Health, United States Public Health Service.

The purpose of the plan is to provide X-ray film packs to be used in the step-wedge method for the assessment of filter needs and also to show beam diameters and collimator needs. Questions to be filled in by the dentists on the outside of the film pack provided data as to the name of the owner, location, type, power ratings, serial number, and model number of the various machines.

The Sur-Pak survey would then be followed by a physical survey of all of the dental X-ray machines in the State, this phase of the program to be carried out by personnel of the Division of Sanitary Engineering, Department of Health and Welfare.

Consequently on March 21, 1962, a request was made by the Maine Division of Dental Health, through the Boston Regional Office, to have Charles J. Gillooly, D.D.S., Chief of the Dental Public Health Services, initiate a Sur-Pak contract. A contract was issued during May, 1962 by the State Assistance Branch, Division of Radiological Health, and on June 19, 1962 the first 100 survey packs were received by the Division of Sanitary Engineering. During the period from June to December, 1962, 350 Sur-Paks were received, and 346 were sent out to dentists in the State. Of the 346 sent out, 308 were returned to the Division of Sanitary Engineering and subsequently sent to the United States Public Health Service, Radiological Health Laboratory in Maryland. Complete tabulated results of the Sur-Pak survey were received from the State Assistance Branch, Division of Radiological Health, and they are incorporated in the body of this report. In addition, a physical survey of all dental X-ray units in the State was completed during the year 1963 by personnel of the Division of Sanitary Engineering, and the results of these surveys are also shown in this report.

The United States Public Health Service, State Assistance Branch, Division of Radiological Health, and the Maine Department of Health and Welfare, Divisions of Dental Health and Sanitary Engineering, wish to thank the Maine Dental Health Association and all of the individual dentists in the State for their splendid cooperation in making this survey a success.

This report was prepared for the practicing dentist. Part I emphasizes the necessity of and the methods for eliminating unnecessary radiation in the dental office. Part II contains a brief tabulation of the results of the combined Sur-Pak and physical survey.

PART I

RADIATION PROTECTION IN DENTAL PRACTICE

Why Should Dentists Be Concerned About Radiation Exposure?

1. Any exposure to X-ray radiation involves some

†Performed for Division of Radiological Health, State Assistance Branch, UNITED STATES PUBLIC HEALTH SERVICE, Washington, D.C. 20201 covering the period June 1, 1962 to December 31, 1963.

*Maine Department of Health and Welfare.

**Division of Radiological Health, United States Public Health Service.

‡Maine Division of Sanitary Engineering, Occupational and Radiological Health Section.

TABLE NO. 1

DENTAL X-RAY UNITS – GENERAL INFORMATION

State Assistance Branch, Division of Radiological Health

Manufacturer	*Model*	*Date of Manufacture*	*Maximum KVP*	*Rating MA*	*Approximate Inherent Filtration mm Al equiv.*	*Focal Spot to Tip of Cone (in.)*	*Added Al (mm) Filtration Recomm.*
Ritter	A	1921-1933	63	10	0.50	7	1.5
	B	1933-1950	65	10	0.50	7	1.5
	D	1950-1956	70	15	0.50	7	1.5
	E	1956-1959	90	15	2.05	7	0.5
	F	1959-	90	15	2.05	7	0.5
General Electric							
Victor	CDX	1923-1933	53	10	0.90	8	1.0
	CDX-E	1933-1954	65	10	1.40	8	0.5
	GE-70	1955	70	10	1.50	8	0.5
	GE-90	1955	90	15	1.50	8	1.0
Weber	5, 6, 7, 7A, 9		65	10	0.50	6.25	1.5
	6R, 11R		70	15	0.50	7	1.5
	90		90	15	1.50	6.25	1.0
	12R		90	15	2.50	7	—
X.R.M.	1B, 2A, & 3A	1939-1957	70	15	0.75	5.5	1.5
	2B, 3B		60	15	2.00	5.5	—
	90		90	15	2.00	5.5	0.5
Philips	A & B		45	5	1.00	4	1.0
Oralix or	C		45	5	2.00	4	—
Norelco	(50) D		50	7	2.00	4	—
Siemens	Heliodent		50	7	2.00	4	—
Wm. Meyers*	A		90	15	0.50	6	2.0
Fischer*	(open-tube)				0.50	—	1.5
	(closed-head)	Discont'd 1952	65	10	0.50	—	1.5
Grenz-Ray*					1.00	7	1.0
Profexray Inc.	MD-2A & MD-3A		60	10	1.70	7.5	0.5
Westinghouse**			65	10	0.70	6.25	1.5
Universal*	(open-tube)				0.75	—	1.5
U-2550	(closed-head)	1955-	63	10	2.00	8	—

*No longer manufacture dental equipment
**Manufactured by X.R.M. for Westinghouse

hazard. The degree of hazard increases as the amount of X-ray exposure increases. Consequently, it is important to keep radiation exposure as low as possible, consistent with diagnostic requirements.

2. Since over half of all X-ray machines are used for dental diagnostic purposes, it is obvious that dentists control a major potential source of radiation exposure to the public.

3. Radiation doses received by patients and dental personnel are sometimes unnecessarily high and may be easily and simply reduced without altering the diagnostic quality of the X-ray films.

What Can The Dentist Do To Reduce Radiation Exposure?

Six relatively simple steps will eliminate the major portion of the unnecessary radiation exposure accompanying the use of X-rays in dental practice. None of the following six steps, if properly carried out, will interfere with, and some will improve, the diagnostic quality of the X-ray films:

1. *Limit the Size of the Useful Beam* – X-rays extending beyond the area of the dental films serve no useful purpose, and for this reason should be eliminated if possible. In most dental X-ray units the installation of a lead washer (collimator) will limit the size of the useful beam. The diameter of the useful beam should not exceed three inches at the tip of the pointer cone, and preferably should not be more than two and three-fourths inches.

Note: Limiting the useful-beam size helps reduce exposure to sensitive areas, such as the lens of the eye, which is outside of the area being examined, and the reduction of the useful beam improves the diagnostic quality of the film, by reducing the amount of fog produced by scattered X-rays.

2. *Filter the Useful Beam* – The useful beam is made up of X-rays of a spectrum of energies. Only the higher-energy X-rays can penetrate the patient's facial tissue and tooth structure, and then react with the emulsion on the film. The X-rays of lower energy are absorbed

TABLE NO. 2

DENTAL ROENTGENOGRAPHIC FILM SPEED-GROUP RATINGS*

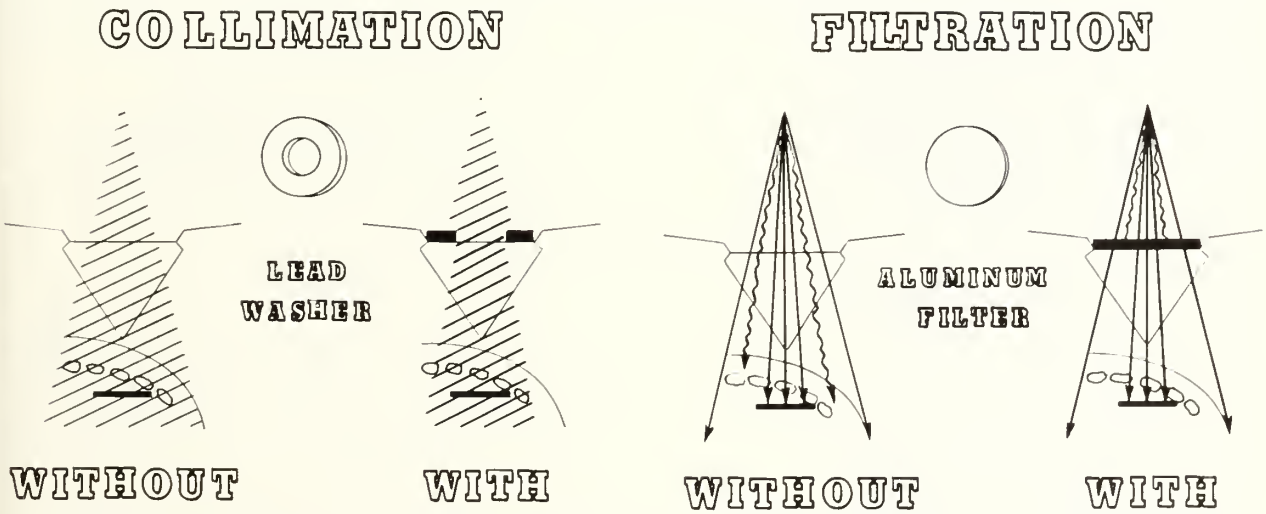
(Data furnished by the manufacturers)

Speed-Group (ASA. Ph 6.1-1961)	*Film Brand*	*Manufacturer or Distributor*
	Periapical Film	
A (slowest)	Rinn DC	Rinn Corp.
B	Dupont "D"	E. I. du Pont de Nemours & Co.
	Kodak Radia-tized	Eastman Kodak Co.
	IFI BH-1	International Films Industries
	Rinn M. F.	Rinn Corp.
C	IFI SBH-1	International Films Industries
	Minimax Intermediate	Minimax Co.
	Rinn E F	Rinn Corp.
D	Dupont "LF"	E. I. du Pont de Nemours & Co.
	Kodak Ultra-Speed	Eastman Kodak Co.
	Minimax Extra-Fast	Minimax Co.
	+ Rinn SF	Rinn Corp.
E (fastest)	Rinn H F	Rinn Corp.
	Bitewing Film	
B	Du Pont "BW"	E. I. du Pont de Nemours & Co.
	Kodak Radia-tized	Eastman Kodak Co.
D	Kodak Ultra-Speed	Eastman Kodak Co.
	Occlusal Film	
D	Kodak Ultra-Speed	Eastman Kodak Co.

*Journal American Dental Association, Vol. 65, pages 280-283

+Manufactured for governmental consumption only

NOTE: There is a two-fold increase in the speed between the groups. This means a dentist using film in B speed-group may use film in speed-group C by simply halving the previously used exposure times.



by the tissue and contribute to the radiation exposure received by the patient. An aluminum filter placed in the path of the useful beam will absorb the low-energy X-rays before they reach the patient. For X-ray units operating up to 70 kvp, the total filtration should be equivalent to 1.5 millimeters of aluminum, and 2.5 millimeters for units operating at higher kilovoltages.

3. *Use the Fastest Film Available* – Exposure to patient and dentist using B speed-group film (see Table No. 2 for Speed-Groups) may be reduced as much as 75% by changing to D speed-group film. Those who have tried fast film in the past without success, will find that today's film is greatly improved. Most X-ray machines using the standard short-pointed cone, and the fastest film (D speed-group) require about one-quarter second of exposure. The ordinary mechanical timer will

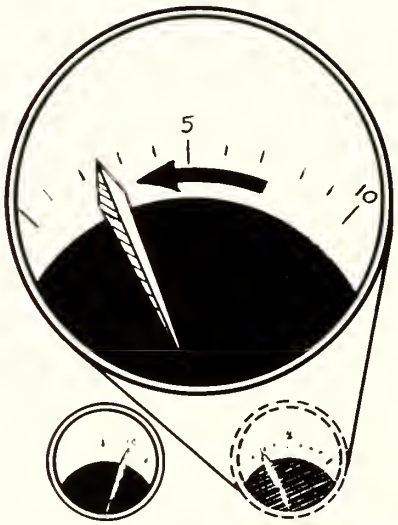
not operate accurately at this exposure time, as a result, many dentists are forced to use exposures of at least one second.

However, there are methods of overcoming this problem, which will permit the use of high-speed film with any unit:

A. The first method is to replace the mechanical timer with an electronic timer, which accurately controls the very short exposure time required by the faster films.

B. If the dentist does not wish to replace his timer, then he may reduce the exposure rate (roentgens per second) at the tip of the cone in order to permit the X-ray machine to be on, one second or more, without overexposure to the faster film. Either of the two following changes will reduce the exposure rate and permit the use of faster films with the mechanical timers:

1. Reduce Milliamperes: The X-ray exposure rate is directly proportional to the milliamperes: Example: a reduction in milliamperes from 10 to 2.5 will reduce the X-ray exposure rate to one-fourth its initial figure, thus permitting an increase in the exposure time from one-quarter of a second to one second. The dentist may make this adjustment himself if the unit has a variable milliampere control, and if it doesn't, this adjustment can be made by an equipment serviceman.
2. Increased Cone Length: As the length of the cone is increased, the exposure rate at its tip decreases. Example: when the focal-film distance is doubled, the exposure rate is reduced one-fourth the original rate. Thus, by replacing a short cone that has a 6″ focal-film distance with a long cone that has a 12″ focal-film distance, the exposure time can be increased from one-quarter second to one second. To obtain the desired film density, a little experimentation may be necessary after making one of the above changes. After a few trial exposures, using an object such as an extracted tooth, the operator should be able to determine the right settings. A patient should not be used for these trial exposures.





REDUCE MILLIAMPERES

4. *Development of Films* – Proper film development helps obtain the best diagnostic quality of the film, and prevents the necessity for retakes. Films should never be overexposed in order to shorten developing time. This practice results in additional radiation exposure to the patient and defeats the purpose of using fast film. For highest film quality and minimum exposure to the patient, the film should be properly exposed and developed for five minutes in a solution maintained at the manufacturer's recommended temperature, usually 68-70° F. If the temperature of the developing solution is not 68° F., an adjustment in the developing time can be made (see table below). It is recommended that a floating thermometer be used to determine the temperature of the developing solution.

MINIMUM DEVELOPING TIME FOR VARIOUS SOLUTION TEMPERATURES

Temperature	*Minimum Time (min.)*
74°F.	3
71°F.	4
70°F.	5
68°F.	6
65°F.	7

Above or below this temperature range, some of the solution chemicals do not function properly. IN ADDITION:

A. Use developers designed for dental X-ray purposes.
B. Regularly change the developer solution.
C. Avoid contamination of the developer solution.
D. Eliminate light leaks in the darkroom.
E. Fix the films for maximum of 10 minutes.
F. Wash for at least 30 minutes and dry thoroughly.
G. Use a safelight that will produce an equivalent amount of light as that produced by a 15-watt bulb with a 6-B Wratten filter at a point 4 feet from the source.

5. *Never Hold the Film, Pointer Cone, or Tube-Hous-*

ing Exposure – Amputation of fingers or hands can result from the unnecessary practice of holding the film or pointer cone during exposure. In the few cases where it may be necessary for the film to be held, as in the case of an uncontrollable child, a member of the patient's family should be utilized, and then only the person's hand should be exposed to the useful beam.

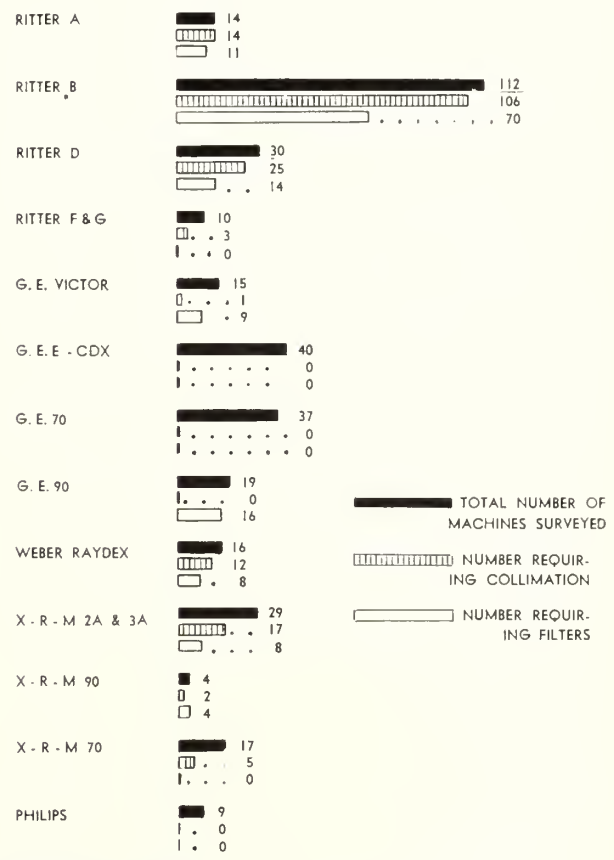
6. *Avoid the Primary and Scattered Radiation* – No one, other than the patient, should be in a position to be exposed to the useful beam. The X-ray operator can easily avoid the useful beam by standing well out of its path. X-rays are scattered in all directions when the X-ray beam strikes the patient's face. The best protection to the operator from scattered radiation is a lead shield. If this is not practicable, the operator should stand as far as possible from the patient, preferably behind a solid wall and at a minimum distance of six feet, which is sufficient for the workloads of the majority of Maine dentists. Any persons not involved in taking X-rays should leave the room and stand behind a solid wall.

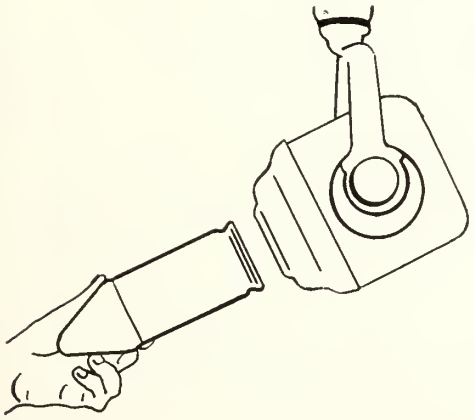
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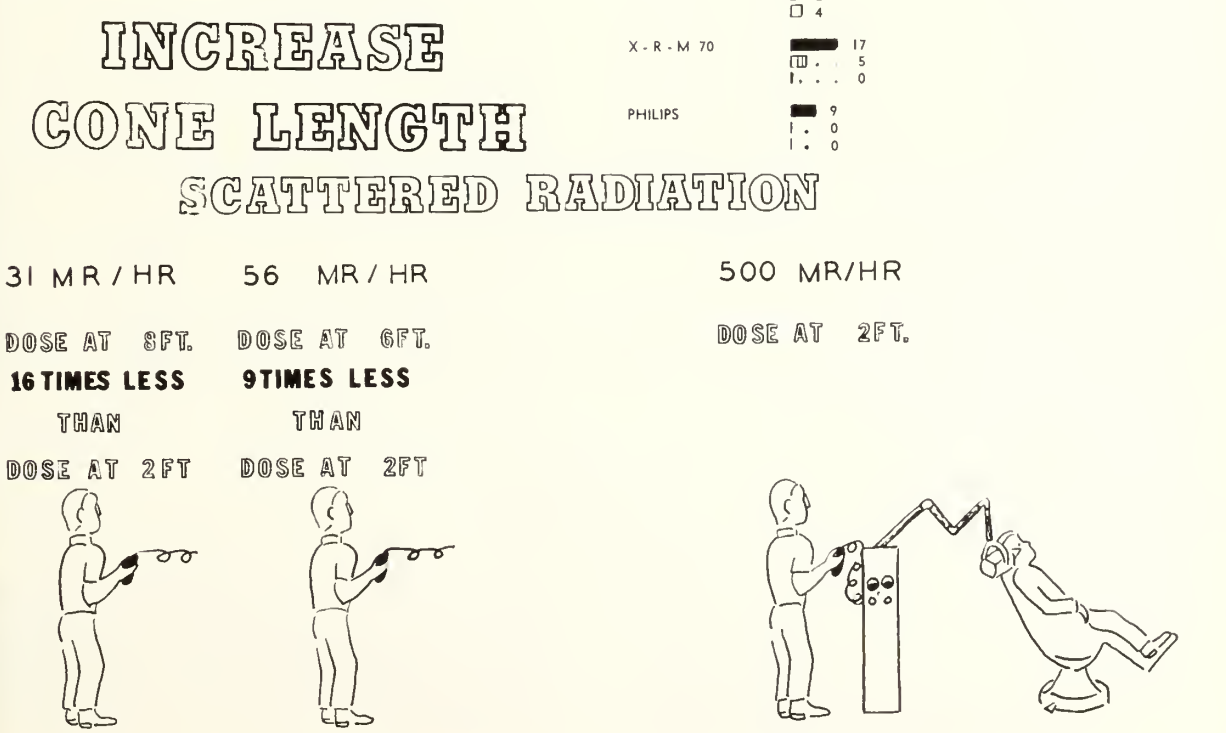
The purpose of the survey was to assess the problems, incorporate protective services and recommend methods of reducing unnecessary ionizing radiation exposure to

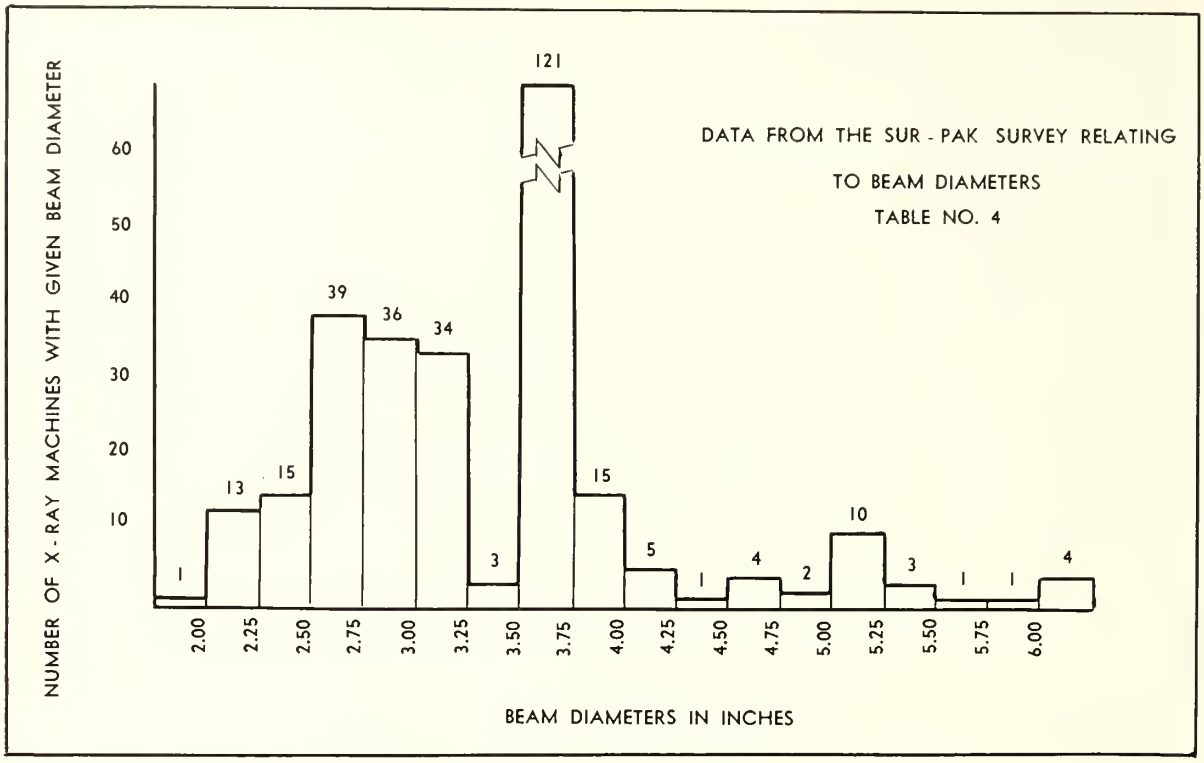
TABLE NO. 3

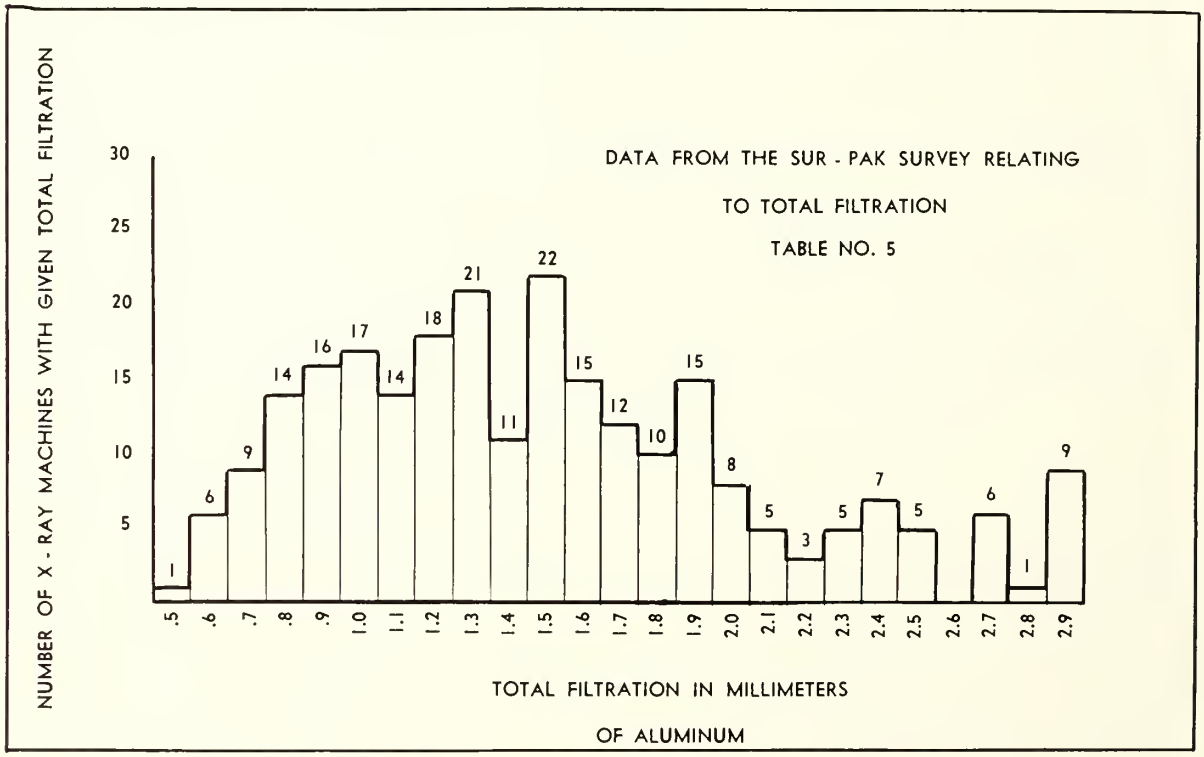
CORRECTION OF X-RAY MACHINES









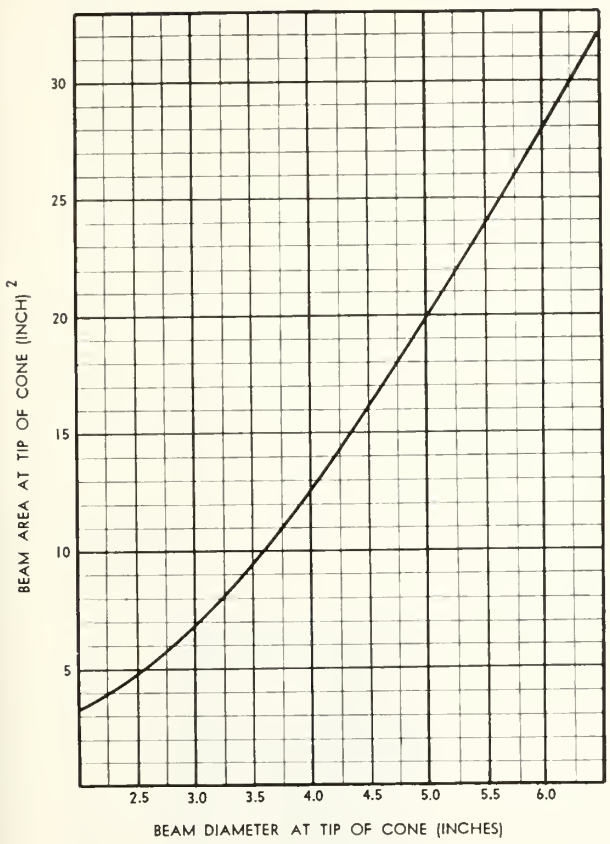


patients and personnel from dental X-rays in the State of Maine.

The Sur-Pak and physical surveys disclosed the following information:

1. *Beam Symmetry* – In 6% of the dental X-ray machines in the State, symmetry of the useful beam was found to be poor and did not approximate a circle or rectangle. The machines with poor beam symmetry were usually those with larger than necessary beam diameters. Therefore, the installation of a lead collimator served not only to decrease the beam size, but to improve the beam symmetry.

TABLE NO. 6

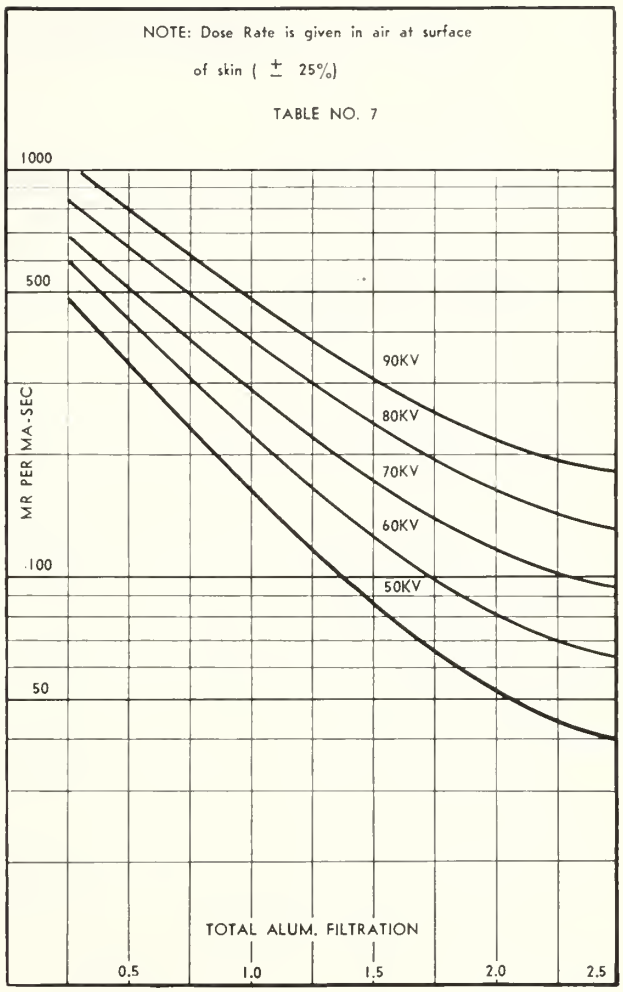


2. *Primary Leakage* – Of the machines surveyed by Sur-Paks, only one was found with primary leakage in the forward direction. This difficulty occurred with an old machine utilizing an exposed leaded-glass tube.

3. *Scattered Radiation on the Films* – Approximately 20% of the Sur-Pak films showed an exposed area with some scattered radiation surrounding it. An additional 7% of the films showed a great deal of scattered radiation surrounding the same area. A study of the data showed no correlation between the amount of scattered radiation and either the type of machine, the total filtration, the collimation, or the operating conditions. It is believed that the governing factor in most cases was the density of the surface on which the Sur-Pak was placed, while being exposed. The denser surfaces reflected more of the useful beam back through the film.

4. *Filtration* – Of the dental X-ray machines in the State, 40% needed additional filtration to increase the total filtration to 1.5 mm Al for 70 kv machines and 2.5 mm Al for 90 kv machines. (For statistics, see Tables No. 3 and 5) An additional millimeter of aluminum added to those machines found to be deficient in filtration, eliminated most of the softer X-rays. These soft X-rays consist of about one-third the original output of the tube. Table No. 7 shows the effect of total filtration on the dose rate.

5. *Collimation* – Approximately 53% of the X-ray



machines were in need of additional collimation to reduce the beam diameter to less than 3 inches at the tip of the cone. (For statistics, see Tables No. 3 and 4) By reducing the beam diameter from 3.60 inches to 2.75 inches, the area of the patient's face exposed to the useful beam is reduced 40%. Table No. 6 shows the relationship between the beam diameter and the area exposed by the beam.

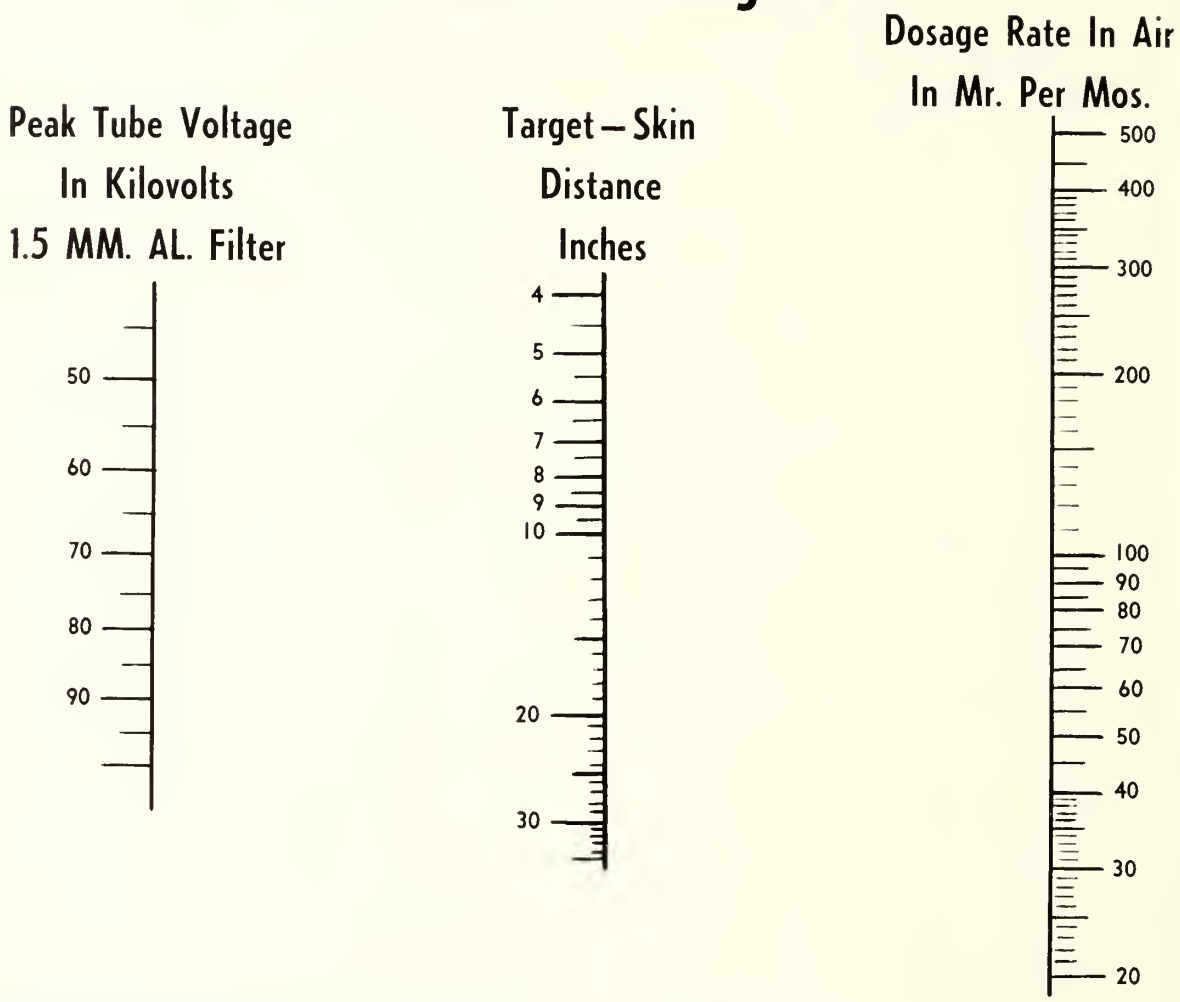
6. *Films Used* – The physical survey disclosed the following percentages of dentists using the various film speed-groups (see Table No. 2).

1% using speed-group A
73% using speed-group B
24% using speed-group C
2% using speed-group D

By employing the methods suggested on page 106 most X-ray machines can be adapted to utilize speed-group D film.

7. *Structural Shielding* – It was found in most cases that ordinary plaster or sheet rock walls provided adequate shielding in dental X-ray rooms. In some circumstances, it was necessary to place additional shielding on the wall separating two adjacent X-ray rooms, to prevent the personnel in one room from being subjected to scat-

Dose Rate Nomogram



tered radiation from the X-ray machine in the next room. In a few cases, because of the limited size of the dental X-ray room, it was necessary to recommend a protective barrier of lead shielding between the dentist and the X-ray machine.

8. *Personnel Monitoring* – The maximum permissible dose (MPD) is defined as the amount of X-ray radiation that, in the light of present knowledge, is not expected to cause appreciable bodily injury to a person at any time during his or her lifetime.

MAXIMUM PERMISSIBLE DOSE*
(To whole body, gonads, blood-forming organs, or lens of eye)

Average Weekly Dose	*Maximum Yearly Dose*	*Maximum Accumulated Dose*
0.1 Roentgen or 100 Milliroentgens	5 Roentgens or 5000 Milliroentgens	5 (N-18**) Roentgens

*Exposure of persons for dental or medical purposes is not counted against their maximum permissible dose.

**N = age in years and is greater than 18. It should also be noted that the occupational limits for individuals under 18 years of age are 10% of those for adults.

9. *Output of Dental X-ray Machines* – For those interested in determining the approximate output of their X-ray machines, a Nomogram has been included.

Dose Rate Nomogram

Example: How to determine the approximate output of a given dental X-ray machine —

Given: Peak tube kilovoltage . 70 kvp
Tube current . . . . 10 ma
Total filtration . . . 1.5 mm aluminum
Target – skin distance* . 8 inches

Find: Dosage rate in air, in roentgens per second

Solution: Place straight edge connecting 70 kvp in first column with 8 inches in second column; read from the third column dosage rate in air = 180 milliroentgens per milliamperes – second (mr/mas)
Exposure time = 1 second
Current = 10 milliamperes
Milliampere – second = 10 mas

Dosage rate in air = (180 mr/mas x 10 mas)
= 1,800 mr/exposure
= 1.8 roentgens per exposure

*Check Table No. 1 for distance from focal spot to tip of cone.

Program

113th Annual Session
Maine Medical Association

June 12, 13, 14, 1966
The Samoset, Rockland

Arranged by the Scientific Committee

RICHARD P. LANEY, M.D., Skowhegan
Chairman

CLEMENT A. HIEBERT, M.D., Portland

WALDO A. CLAPP, M.D., Lewiston



Dr. Laney

Sponsors:

The Scientific Program for the annual session of the Maine Medical Association in cooperation with the Technical Exhibitors and the Council of the Maine Medical Association is made possible this year by grants from:

Merck Sharp & Dohme Postgraduate Program
West Point, Pennsylvania

Eli Lilly and Company State Medical Convention Program
Indianapolis, Indiana

Diabetes Detection Program of the State of Maine Department of Health and Welfare
Augusta, Maine

Warner-Chilcott Laboratories
Morris Plains, New Jersey

For this cooperation and support the members of the Scientific Committee are grateful.

Information

Registration:

Registration throughout the session will be in the Lobby at The Samoset. Registration fee $1.00.

Sunday	June 12 — 9:00 A.M. to 5:30 P.M.
Monday	June 13 — 9:00 A.M. to 6:00 P.M.
Tuesday	June 14 — 9:00 A.M. to 4:30 P.M.

Telephone: The number at The Samoset is Rockland, 594-8411.

Visiting Delegates:

Introduction of Visiting Delegates will take place at meetings of the House of Delegates on Sunday, June 12 or at the General Assembly, Monday afternoon, June 13.

Scientific and Special Exhibits:

These exhibits are listed on page 119.

Technical Exhibits:

The forty-three companies that contribute to the program by participation in the Technical Exhibits are listed on pages 119 through 120. You will pass these exhibits on your way through the Lobby en route to the Ballroom (where the Scientific Sessions will be held) to the Dining Room or to the Golf Course. Please show your appreciation by visiting these exhibits at every possible opportunity.

Sunday, June 12

9:30 A.M. First Meeting of the House of Delegates

Call to order: GEORGE E. SULLIVAN, M.D., President-Elect

Presiding: Speaker of the House, LINUS J. STITHAM, M.D.

Presentation of A. H. Robins' Physician Award for Community Service

Presentation of Parke, Davis & Company Historical Paintings to John F. Dougherty, M.D., President, Maine Medical Association

12:30 P.M. Luncheon

3:00 P.M. Second Meeting of the House of Delegates

6:30 P.M. Dinner

Speaker: WILLIAM C. MOTT, Rear Admiral, USN (Ret.), Executive Vice President, United States Independent Telephone Association, Washington, D.C.

Subject: **The Doctor as a Revolutionary**

Monday, June 13

Scientific Program

Scientific speakers are sponsored in part by a grant from the Merck Sharp & Dohme Postgraduate Program.

9:00 A.M. to 12:00 NOON

Presiding – RICHARD P. LANEY, M.D.

9:00 A.M. **Film – Roentgen Anatomy of the Normal Heart**

FRANCIS A. SPELLMAN, M.D., Togus, Maine in honor of Centennial Year of the Veterans Administration Center, Togus

9:30 A.M. **Diseases of the Esophagus**

JULIUS WOLF, M.D., Chief, Medical Service, Veterans Administration Hospital, Bronx, New York

10:30 A.M. **Panel – Management of Biliary Tract Disease and Associated Conditions**

Moderator: GEORGE J. ROBERTSON, M.D., Medical Director, Bingham Associates Fund and Senior Physician in Internal Medicine, Tufts-New England Medical Center, Boston, Massachusetts

PERRY J. CULVER, M.D., Associate Physician, Massachusetts General Hospital, Boston, Massachusetts

KENNETH W. WARREN, M.D., Department of General Surgery, Lahey Clinic Foundation, Boston, Massachusetts

ROBERT E. WISE, M.D., Chairman, Department of Diagnostic Radiology, Lahey Clinic Foundation, Boston, Massachusetts

12:00 NOON to 2:00 P.M. Luncheon

Scientific Program

1:30 to 4:00 P.M.

Presiding – CLEMENT A. HIEBERT, M.D.

Sponsored by the Maine Chapter, American College of Surgeons and Maine Trauma Committee

1:30 P.M. Business Meeting, Maine Chapter American College of Surgeons

2:00 P.M. **Pain, Psychosomatics, and the Pancreas**

GEORGE L. NARDI, M.D., Visiting Surgeon, Massachusetts General Hospital and Associate Clinical Professor of Surgery, Harvard Medical School, Boston, Massachusetts

3:00 P.M. **Prevention and Treatment of Low Back Pain**

HANS KRAUS, M.D., Associate Professor of Physical Medicine and Rehabilitation, New York University School of Medicine, New York, New York

4:00 P.M. **General Assembly**

Presiding, JOHN F. DOUGHERTY, M.D., President

President's Address

Election of President-elect

Presentation of the Mead Johnson Laboratories Aesculapius Award for Outstanding Exhibit

6:00 to 7:00 P.M. Social Hour, Dutch Treat, Ballroom

7:00 P.M. **Annual Banquet**

GOVERNOR JOHN H. REED, Guest of Honor

Presentation of Honorary Pins

Speaker: HOMER H. STRYKER, M.D., President, Stryker Corporation, Kalamazoo, Michigan

Subject: **Backing Into Business Without Even Trying**

Tuesday, June 14

Scientific Program

9:30 A.M. to 12:00 NOON

Presiding – WALDO A. CLAPP, M.D.

9:30 A.M. **New Concepts in the Diagnosis and Prevention of Diabetes**

LEO P. KRALL, M.D., Senior Staff, Joslin Clinic and New England Deaconess Hospital, Boston, Massachusetts

Sponsored by the Diabetes Detection Program of the State of Maine Department of Health and Welfare in conjunction with the Maine Chapter, American Academy of General Practice.

10:30 A.M. **Panel – Medical Emergencies**

Moderator: GEORGE W. WOOD, III, M.D., Internist, Brewer

Burns and Peripheral Emboli

LLOYD BROWN, M.D., Surgeon, Bangor

Hypoxia

WARREN G. STROUT, M.D., Anesthesiologist, Bangor

Shock

EDWARD B. BABCOCK, M.D., Internist, Bangor

The Unconscious Patient

HAROLD D. CROSS, M.D., General Practitioner, Hampden Highlands

12:00 NOON to 2:00 P.M. Luncheon

Scientific Program

2:00 P.M.

Presiding – RICHARD P. LANEY, M.D.

Sponsored by the Maine Medico-Legal Society

2:00 P.M. **Coordination and Procedure in the Boston Strangler Cases**

MR. JOHN S. BOTTOMLY, Former Assistant Attorney General of Massachusetts, Boston, Massachusetts

6:30 P.M. Lobster Bake

Presentation of Golf Prizes by

DANIEL R. SHIELDS, M.D., Lewiston, Chairman, Golf Tournament

Specialty Group Meetings

Monday, June 13

Section on Ophthalmology of the M.M.A.

DEXTER J. CLOUGH, 2ND, M.D., Bangor, presiding

Clinical Fundus Pathology

DAVID D. DONALDSON, M.D., Boston, Massachusetts

Maine Society of Clinical Hypnosis

ROBERT F. RUSSELL, M.D., Penobscot, presiding

Practical Applications of Hypnosis in General Medicine

HERBERT SPIEGEL, M.D., Assistant Clinical Professor of Psychiatry, College of Physicians and Surgeons, Columbia University, New York, New York

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Maine Thoracic Society, Maine Society of Internal Medicine and Maine Society of Anesthesiology

2:00 P.M.

RUDOLPH HAAS, M.D., Lewiston, presiding

Pre and Postoperative Care of Patients With Chronic Lung Disease

LEROY D. VANDAM, M.D., Clinical Professor of Anesthesia, Harvard Medical School, Boston, Massachusetts

3:00 P.M.

RICHARD S. HAWKES, M.D., Portland, presiding

Utilization, How Useful?

FRANK P. FOSTER, M.D., Department of Internal Medicine, Lahey Clinic Foundation, Boston, Massachusetts

Tuesday, June 14

10:00 A.M. Maine Medico-Legal Society

Business Meeting

MR. GEORGE C. WEST, Deputy Attorney General of Maine, Augusta, presiding

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12:00 NOON Maine Radiological Society
Luncheon Meeting

CHARLES W. CAPRON, M.D., Portland, presiding

Speaker: EDWARD B. D. NEUHAUSER, M.D., Radiologist-in-Chief, Children's Medical Center, Boston, Massachusetts

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2:00 to 4:00 P.M.

Maine Society of Obstetrics and Gynecology

Business Meeting

FREDERICK B. LIDSTONE, M.D., Auburn, presiding

Ear, Nose and Throat Group

JOHN E. WHITWORTH, M.D., Bangor, presiding

Pain in the Head

RUDOLPH W. PEARSON, M.D., Chief of the Department of Otolaryngology, Rhode Island Hospital, Providence, Rhode Island

Luncheon Meetings

12:00 NOON

Monday, June 13

Orthopedists (Knox Room)

Tuesday, June 14

Amy W. Pinkham Fund Committee (Main Dining Room)

Maine Radiological Society (Penobscot Room)

SPECIAL NOTICES

Election of President-elect

The election of a President-elect will take place at the General Assembly, June 13 at 4:00 p.m.

Election of Councilors

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 12 at 3:00 p.m.

First District – Cumberland and York

Second District – Androscoggin, Franklin and Oxford

In accordance with the By-Laws, 'N'ominations for members of the Council for any District where there is a vacancy shall be made by a caucus of the members of the House of Delegates of that District. Each candidate for Councilor must be a resident of the District for which he is nominated."

Council Meetings

The Council will meet on Saturday, June 11 and daily throughout the session at a time and place to be announced.

Committee Meetings

Amy W. Pinkham Fund Committee, Luncheon Meeting, Tuesday, June 14.

Committee on Recruitment, Aid and Placement Tuesday, June 14 – 2:30 p.m.

Technical Exhibits

Please visit these exhibits.

Golf Tournament

Daniel R. Shields, M.D., Lewiston, Chairman

HONORARY PINS

Presentation of the Association's Honorary Pins will be made by John F. Dougherty, M.D., President of the M.M.A., at the Annual Banquet, Monday evening, June 13 at 7:00 P.M.

FIFTY-YEAR PINS

Fifty-Year Lapel Pins will be presented to the following members who were graduated from Medical School in 1916:

Cumberland County
George O. Cummings, Sr., M.D., Portland
Bowdoin Medical School

Herman C. Petterson, M.D., Chebeague Island
Hahnemann Medical College

Hancock County
Harold S. Babcock, M.D., Castine
Jefferson Medical College

Somerset County
Maurice E. Lord, M.D., Lake Placid, Florida
University of Vermont College of Medicine

FIFTY-FIVE-YEAR PINS

Fifty-Five-Year Pins will be presented to the following members who received Fifty-Year Pins in 1961:

Cumberland County
James Patterson, M.D., South Portland
Rush Medical College

Knox County
Fred G. Campbell, M.D., Warren
Baltimore Medical College

Lincoln-Sagadahoc County
Rufus E. Stetson, M.D., Damariscotta
Columbia University College of Physicians and Surgeons

Oxford County
Lester Adams, M.D., Thomaston
Johns-Hopkins University School of Medicine

Somerset County
Merlon A. Webber, M.D., Pittsfield
Bowdoin Medical School (1910)

Waldo County
Carl H. Stevens, M.D., Belfast
Bowdoin Medical School

SIXTY-FIVE-YEAR PINS

A Sixty-Five-Year Pin will be presented to the following member who received his Fifty-Year Pin in 1951:

York County
Ansel S. Davis, M.D., Springvale
Bowdoin Medical School

Visiting Delegates

The Connecticut State Medical Society
CHARLES N. BARBOUR, M.D., West Hartford
SYDNEY LURIA, M.D., Bridgeport

The Massachusetts Medical Society
DONALD A. NICKERSON, M.D., Salem

The New Hampshire Medical Society
PETER T. LAMPESIS, M.D., Dover

Medical Society of the State of New York
THURMAN B. GIVAN, M.D., Brooklyn

The Rhode Island Medical Society
HANNIBAL HAMLIN, M.D., Providence

Vermont State Medical Society
B. ALBERT RING, M.D., Burlington

Delegates to Out-of-State Meetings

The Connecticut State Medical Society
ROBERT M. KNOWLES, M.D., Portland

The Massachusetts Medical Society
HARLAND G. TURNER, M.D., Norridgewock

The New Brunswick Medical Society
HAZEN C. MITCHELL, M.D., Calais

The New Hampshire and Vermont State Medical Societies
PAUL S. HILL, JR., M.D., Saco

Medical Society of the State of New York
JOHN F. DOUGHERTY, M.D., Bath

The Rhode Island Medical Society
LEONARD G. MIRAGLIUOLO, M.D., Bangor

County Delegates

FIRST DISTRICT

Cumberland County Medical Society
Delegates: Stanley B. Sylvester, M.D., Union Mutual Life Insurance Co., 400 Congress St., Portland, Secretary
(2 years)
Charles R. Glassmire, M.D., 37 Deering St., Portland
Lloyd G. Davies, M.D., 78 Main St., Fryeburg
Alvin A. Morrison, M.D., 57 Deering St., Portland
Frederick B. Clark, M.D., 131 State St., Portland
(1 year)
Charles R. Geer, M.D., 208 Vaughan St., Portland
Howard P. Sawyer, Jr., M.D., 22 Bramhall St., Portland
Maurice Van Lonkhuyzen, M.D., 131 State St., Portland
Philip P. Thompson, Jr., M.D., 131 Chadwick St., Portland
David K. Lovely, M.D., 46 Deering St., Portland
Clement A. Hiebert, M.D., 18 Bramhall St., Portland

Alternates
(2 years)
Sidney R. Branson, M.D., 37 Main St., South Windham
Kirk M. Barnes, M.D., Baribeau Dr., Brunswick
John F. Gibbons, M.D., 22 Bramhall St., Portland
Robinson L. Bidwell, M.D., 31 Bramhall St., Portland
(1 year)
Hugh P. Robinson, M.D., 27 Deering St., Portland
Raphael F. Turgeon, M.D., 367 Main St., Westbrook
Harold N. Burnham, M.D., 130 Main St., Gorham
Irving J. Poliner, M.D., 235 State St., Portland
Louis A. Ciampi, M.D., 326 Stevens Ave., Portland

York County Medical Society
Delegates: Charles W. Kinghorn, M.D., 4 Wentworth St., Kittery, Secretary
Carl E. Richards, M.D., 34 Winter St., Sanford
Robert F. Ficker, M.D., Maine St., Kennebunkport
Thomas Anton, M.D., 260 Main St., Biddeford

Alternates
Irvin Dorfman, M.D., Webber Hospital, Biddeford
Stephen A. Cobb, M.D., 34 Winter St., Sanford
Kenneth E. Leigh, M.D., Brixham Rd., York

SECOND DISTRICT

Androscoggin County Medical Association
Delegates: Donald L. Anderson, M.D., 369 Main St., Lewiston, Secretary
Henry C. Thacher, M.D., 117 Goff St., Auburn
Edward L. Reeves, M.D., 179 Sabattus St., Lewiston
Morris E. Goldman, M.D., 185 Webster St., Lewiston
Joseph J. Rando, M.D., 111 Webster St., Lewiston

Alternates
Harvey J. Proulx, M.D., 92 Pine St., Lewiston
Frederick B. Lidstone, M.D., 117 Goff St., Auburn
Charles A. Hannigan, M.D., 85 Goff St., Auburn
Philip L. Archambault, M.D., 346 Main St., Lewiston

Franklin County Medical Society
Delegates: Maynard B. Colley, M.D., 114 Main St., Farmington, Secretary
Hays G. Browne, M.D., 9A Main St., Farmington

Alternate
Paul A. Brinkman, M.D., Farmington

Oxford County Medical Society
Delegates: Ake Akerberg, M.D., 2 E. Main St., South Paris, Secretary
James A. MacDougall, M.D., 303 Penobscot St., Rumford
Peter B. Aucoin, M.D., 151 Franklin St., Rumford

Alternates
Albert P. Royal, Jr., M.D., 82 Maine Ave., Rumford
Joelle C. Hiebert, Jr., M.D., Box 148, Norway

THIRD DISTRICT

Knox County Medical Society
Delegates: William W. Ward, M.D., P.O. Box 804, Rockland, Secretary
Johan Brouwer, M.D., 5 Beech St., Rockland
Thomas W. Williams, M.D., 22 White St., Rockland
Continued on Page 118

Pro-Banthine
(propantheline bromide)

Intragastric photography has provided a new and precise method of measuring the effectiveness of anticholinergic drugs. The transition from gastric motor activity to relaxation seen with effective doses of such drugs takes only a few seconds and is easily demonstrated.

The importance of vagal stimulation of gastric hyperacidity and hypermotility makes such measurements particularly important in evaluating the parasympatholytic effect of drugs used in patients with peptic ulcer, gastritis, biliary dyskinesia and other gastrointestinal disorders.

See fo

Pro-Banthine has been shown[1] to produce complete gastric motor inactivity with doses of 6 to 8 mg. intravenously. Comparison tests were made with the belladonna fraction, atropine. Measured usual dosage unit versus usual dosage unit, Pro-Banthine was more than four times as effective as the belladonna alkaloid.

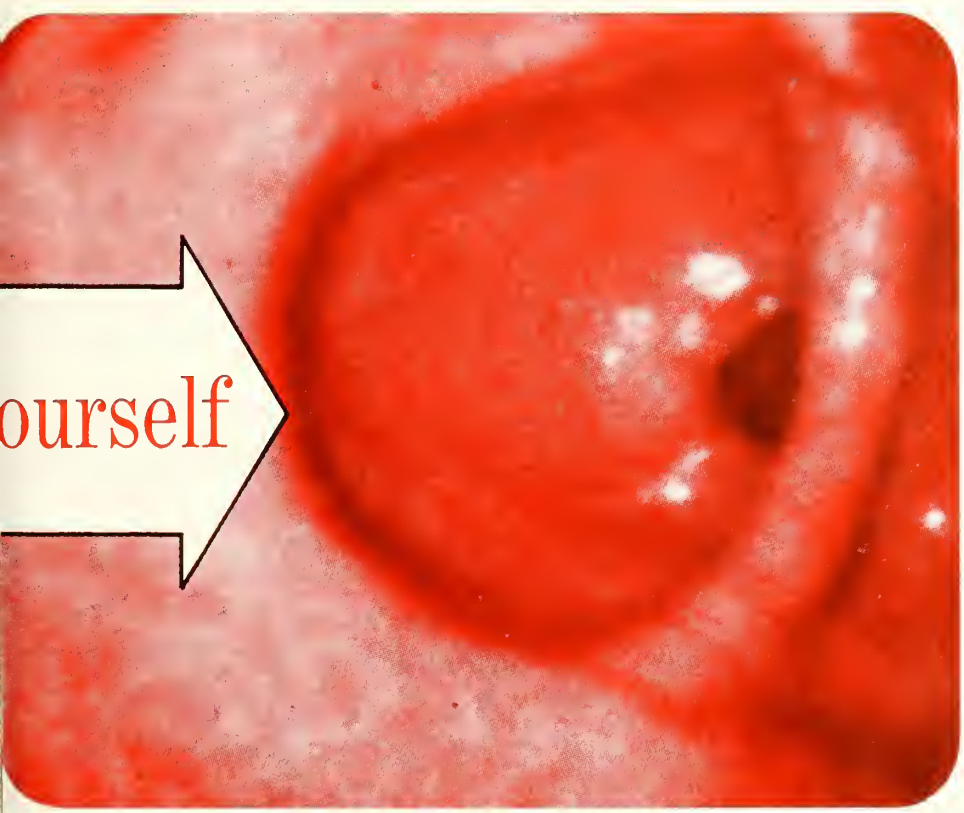
Indications: Peptic ulcer, functional hypermotility, irritable colon, pylorospasm and biliary dyskinesia.

Oral Dosage: Adequate dosage should be given for optimal results. For most *adult* patients this will be four to six 15-mg. tablets daily in divided doses. In severe conditions as many as two tablets four to six times daily may be required. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg., as prolonged-acting tablets of 30 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

Side Effects and Contraindications: Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. Pro-Banthine is contraindicated in patients with glaucoma, severe cardiac disease and prostatic hypertrophy.

1. Barowsky, H.; Greene, L., and Paulo, D.: Cinegastroscopic Observations on the Effect of Anticholinergic and Related Drugs on Gastric and Pyloric Motor Activity, Amer. J. Dig. Dis. *10*:506-513 (June) 1965.

s Effective



Complete gastric relaxation with Pro-Banthine. As this intragastric photograph demonstrates, gastric relaxation is attained with 6 mg. of Pro-Banthine intravenously; the antrum is relaxed and the pyloric orifice remains open. Full intravenous doses of atropine (4 mg.) produce no measurable effect.

SEARLE

Research in the Service of Medicine

COUNTY DELEGATES – *Continued from Page 115*

Alternate
Wesley N. Wasgatt, M.D., 41 Talbot Ave., Rockland

Lincoln-Sagadahoc County Medical Society
Delegates: George W. Bostwick, M.D., P.O. Box 388, Newcastle, Secretary
John F. Andrews, M.D., 20 West St., Boothbay Harbor
Ralph C. Powell, M.D., Damariscotta

Alternates
Charles E. Burden, M.D., 1 North St., Bath
Paul A. Fichtner, M.D., 781 High St., Bath

FOURTH DISTRICT

Kennebec County Medical Association
Delegates: Lucien F. Veilleux, M.D., 30 Elm St., Waterville, Secretary
John M. H. Barnard, M.D., Doctors Park, 89 Hospital St., Augusta
Earle M. Davis, M.D., 2 School St., Waterville
Paul A. Jones, Jr., M.D., 2 School St., Waterville
Valentine J. Moore, M.D., Thayer Hospital, Waterville
Francis J. O'Connor, M.D., 4 Woodlawn St., Augusta

Alternates
Richard T. Chamberlin, M.D., 14 Gilman St., Waterville
Richard L. Chasse, M.D., 18 Park St., Waterville
Theodore M. Russell, M.D., 20 Western Ave., Augusta
John H. Shaw, M.D., 131 Sewall St., Augusta
Francis A. Spellman, M.D., Veterans Adm. Ctr., Togus

Somerset County Medical Society
Delegates: John P. Dow, M.D., 59 Main St., Pittsfield, Secretary
Richard P. Laney, M.D., 50 Water St., Skowhegan
H. Carl Amrein, M.D., 29 Weston Ave., Madison

Alternates
Harland G. Turner, M.D., Box 38, Norridgewock
Edgar J. Smith, M.D., 1 Park St., Fairfield

Waldo County Medical Society
Delegates: Seth H. Read, M.D., 15 Church St., Belfast, Secretary
Norman E. Cobb, M.D., 132 Main St., Belfast

Alternate
George L. Temple, M.D., Fahey St., Belfast

FIFTH DISTRICT

Hancock County Medical Society
Delegates: Russell G. Williamson, M.D., Blue Hill Mem. Hospital, Blue Hill, Secretary
Herbert T. Wilbur, Jr., M.D., 100 Main St., Southwest Harbor
Russell M. Lane, M.D., P.O. Box "J", Blue Hill

Alternates
Arthur M. Joost, Jr., M.D., P.O. Box 502, Bucksport
Elizabeth E. Williamson, M.D., Blue Hill

Washington County Medical Society
Delegates: Karl V. Larson, M.D., East Machias, Secretary
Hazen C. Mitchell, M.D., Calais

Alternate
James C. Bates, M.D., Eastport

SIXTH DISTRICT

Aroostook County Medical Society
Delegates: George J. Harrison, M.D., Market Square, Houlton, Secretary
Melvin R. Aungst, M.D., Morneault Bldg., Fort Kent
John B. Madigan, M.D., Houlton
Raymond G. Giberson, M.D., 156A Academy St., Presque Isle

Alternates
Rodrigue J. Albert, M.D., 77 Main St., Fort Kent
George F. Higgins, M.D., 122 Academy St., Presque Isle
H. Douglas Collins, M.D., Caribou Clinic, Caribou

Penobscot County Medical Association
Delegates: Charles D. McEvoy, Jr., M.D., 186 State St., Bangor, Secretary
George O. Chase, M.D., Eastern Maine Gen. Hospital, Bangor
James D. Clement, Jr., M.D., 77 Essex St., Bangor
James E. Hamlin, M.D., Main St., E. Millinocket
Leonard G. Miragliuolo, M.D., 10 Maple St., Bangor
Richard V. Duffey, M.D., 187 N. Main St., Brewer

Alternates
John S. Houlihan, M.D., 209 State St., Bangor
Richard A. Gaillard, M.D., 276 State St., Bangor
William M. Shubert, M.D., 317 State St., Bangor
Clement S. Dwyer, M.D., 205 French St., Bangor
John C. Bjorn, M.D., Hampden Highlands

Piscataquis County Medical Society
Delegates: Isaac Nelson, M.D., Box 336, Greenville, Secretary
Linus J. Stitham, M.D., 50 Main St., Dover-Foxcroft

Alternate
Charles H. Lightbody, M.D., North Main St., Guilford

Scientific and Special Exhibits

AMA Department of Drugs – "Is This Drug Safe?"

Associated Hospital Service of Maine

Boston Medical Reports

Cervical Cancer Detection Program Film – Me. Chap. AAGP

Department of HEW – Food and Drug Adm., Washington, D.C. – "FDA's Adverse Reaction Program"

Division of Cancer Control of the State of Maine Department of Health and Welfare

Division of Eye Care and Special Services of the State of Maine Department of Health and Welfare

"Dose Response: Key to Effective Treatment" – (Exhibit presented by Fred J. Phillips, M.D.)

"Hunting in Maine – Surgical Aspects" – (Exhibit presented by Clement A. Hiebert, M.D.)

Maine Cancer Society

Maine Chapter, American Academy of General Practice

Maine Dairy Council

Maine Heart Association, Inc.

Maine Society of Anesthesiology

Maine Society of Clinical Hypnosis

Maine Radiological Society

Maine Thoracic Society

New England Physicians Retirement Program

Pernicious Anemia Detection Program, Eastern Maine General Hospital – "Folic Acid Deficiency in Pregnancy"

Pineland Hospital and Training Center – "Tuberous Sclerosis"

Thayer Hospital – "The Synthesis of Health"

The National Foundation – "Chemistry, Chromosomes and Congenital Anomalies"

"The Rising Problem of Motorcycle Accidents" (Exhibit presented by Richard C. Dillihunt, M.D.)

"Today's Problems of Diuretic Therapy" – (Exhibit presented by Arthur Bernstein, M.D.)

Veterans Administration Center, Togus, Maine – "Current Concepts of the Urethral Sphincter and Dynamic Sphincterometer Studies"

Vocational Rehabilitation Division of the State of Maine Department of Education

Mead Johnson Laboratories Aesculapius Award for Outstanding Scientific Exhibit

With the aim of supporting and encouraging the development of scientific exhibits of high quality, Mead Johnson Laboratories has created the Aesculapius Award, designed to recognize and reward excellence in concept, originality and execution of scientific exhibit presentations at the Annual Session of the M.M.A.

Under the terms of this program, the most outstanding scientific exhibit will be selected by a committee of the medical society. The author of the winning exhibit will be presented with an appropriate certificate and a cash prize of $200.

This award will be presented at the General Assembly on Monday, June 13 at 4:00 p.m.

Technical Exhibits

Abbott Laboratories, North Chicago, Illinois
Representative: Mr. P. F. Woodlock

The Alkalol Company, Taunton, Massachusetts
Representative: Mr. E. W. LeClair

Americana Corporation, 100 Cabot St., Needham Heights, Massachusetts

Ayerst Laboratories, 245 Paterson Ave., Little Falls, New Jersey
Representative: Mr. Edward McMahon

Bicknell Photo Service Inc., 24 Free St., Portland, Maine
Representatives: Mr. Bud Millman and Mr. Fred Wormell

Elmer N. Blackwell, Surgical Appliance Specialist, 565 Congress St., Room 207, Portland, Maine
Representatives: Mr. Elmer N. Blackwell and Mr. Oakley R. Sanborn

The Borden Company, 350 Madison Ave., New York, New York
Representative: Mr. Joseph R. Galvin

Brewer & Company Inc., 67 Union St., Worcester, Massachusetts
Representative: Mr. Walter L. Spaulding, Jr.

Bristol Laboratories, Syracuse, New York
Representative: Mr. William Read

Burroughs Wellcome & Co. (U.S.A.) Inc., 1 Scarsdale

Rd., Tuckahoe, New York
Representatives: Mr. R. D. St. Laurent, Mr. L. F. Grillo and Mr. E. J. Benoit

Ciba Pharmaceutical Company, Summit, New Jersey
Representative: Mr John H. Angis

The Coca-Cola Company, P. O. Drawer 1734, Atlanta, Georgia

Encyclopaedia Britannica, 425 North Michigan Ave., Chicago, Illinois
Representative: Mr. J. Quinn

Endo Laboratories Inc., 1000 Stewart Ave., Garden City, New York
Representatives: Mr. John Winfield and Mr. David D. Green

Geo. C. Frye Co., 685 Congress St., Portland, Maine
Representatives: Mr. Milton S. Kimball, Mr. John F. Kimball, Mr. Sidney F. Cheney, Mr. Robert S. Cheney, Mr. Douglas L. Cyr, Mr. Arthur R. Wickham and Mr. Robert L. May

Geigy Pharmaceuticals, P. O. Box 430, Yonkers, New York
Representatives: Mr. George Williams and Mr. Peter Wilkinson

Lederle Laboratories, Pearl River, New York
Representatives: Mr. R. Maffei, Mr. J. Boucher and Mr. R. G. Pelletier

Maine Surgical Supply Co., 233 Vaughan St., Portland, Maine
Representatives: Mr. Robert Axelsen, Mr. Lawrence Gardiner, Mr. Philip Dana, Jr., Mr. John H. Lacy, Mr. George H. Munroe and Mr. Louis Olore

Mallard Incorporated, 3021 Wabash Ave., Detroit, Michigan
Representative: Mr. Roland Stickney

McNeil Laboratories, Inc., Camp Hill Rd., Fort Washington, Pennsylvania
Representative: Mr. Joseph A. Ruest, Jr.

Mead Johnson Laboratories, Evansville, Indiana
Representatives: Mr. Jack J. Haskins and Mr. John G. Kubin

Medical Plastics Laboratory, Inc., Gatesville, Texas
Representative: Mr. Emanuel R. Goldman

Merck Sharp & Dohme, West Point, Pennsylvania
Representatives: Mr. Robert S. Baxendale and Mr. David Vincent

Witold J. Mikelk, 11 Field St., Auburn, Massachusetts

Parke, Davis & Company, Joseph Campau Avenue at the River, Detroit, Michigan
Representatives: Mr. L. Bosworth and Mr. M. Dole

A. H. Robins Company, Inc., 1407 Cummings Dr., Richmond, Virginia
Representative: Mr. Henry N. Paradis, Jr.

Roche Laboratories, Nutley, New Jersey
Representative: Mr. Brad Garcelon

J. B. Roerig & Company, 230 Brighton Rd., Clifton, New Jersey
Representatives: Mr. C. J. (Tom) Johnson and Mr. Carl Williams

William H. Rorer, Inc., Fort Washington, Pennsylvania
Representatives: Mr. J. M. Beward, Mr. R. W. Roffler and Mr. R. A. Gelinas

Ross Laboratories, Columbus, Ohio
Representatives: Mr. H. Hutchinson, Jr. and Mr. R. Kaufman

Sandoz Pharmaceuticals, Hanover, New Jersey
Representative: Mr. Herman Emidy

Schering Corporation, 1011 Morris Ave., Union, New Jersey
Representatives: Mr. Jack Arlaud and Mr. Floyd Selby

Sealy Mattress Company, 38-42 Everett St., Allston, Massachusetts
Representative: Mr. Henry Gorman

G. D. Searle & Co., P. O. Box 5110, Chicago, Illinois
Representatives: Mr. Allison Zahniser and Mr. A. L. Grimes

Smith Kline & French Laboratories, 1500 Spring Garden St., Philadelphia, Pennsylvania
Representative: Mr. I. Bruce Greene

Smith, Miller & Patch, Inc., 902 Broadway, New York, New York
Representatives: Mr. Kenneth Mullen and Mr. Frederick Murphy

E. R. Squibb & Sons, 745 Fifth Ave., New York, New York

Stimpson Copy Products, Inc., 652 Congress St., Portland, Maine
Representatives: Mr. Sidney Schuman and Mr. Donald Thompson

The Stuart Company, 3360 East Foothill Blvd., Pasadena, California
Representatives: Mr. Percy Valley and Mr. Harry Reinhorn

Surgeons' and Physicians' Supply Co., 961 Commonwealth Ave., Boston, Massachusetts
Representatives: Mr. Lester Clough, Mr. Irving Beers and Mr. John Stutz

Syntex Laboratories, Inc., Stanford Industrial Pk., Palo Alto, California
Representatives: Mr. J. Perroni and Mr. D. Mahoney

U. S. Vitamin & Pharmaceutical Corporation, 800 Second Ave., New York, New York

The Upjohn Company, Kalamazoo, Michigan

State Of Maine

Department of Health and Welfare

DEAN H. FISHER, M.D.
COMMISSIONER

Public Health in Transition—Part II*

HARRY ALLEN, M.D.**

As a result of the Federal Health Insurance Program, somewhere in the vicinity of 19 million people in the United States will be eligible for minimal home health services which by definition must include nursing care and one other modality of therapy. This could be physical therapy, occupational therapy, speech therapy, medical social work and in some cases the service of interns and residents. *Currently, in many parts of the country, these services are nonexistent, and yet on July 1, 1966 this is a legal benefit of the Social Security beneficiaries.*

The situation in New England is rather unique. Despite the fact that New England provided leadership in the organization of local health services, today one is hard put to list over 20 competent, fully staffed units. Lack of legislative support and political traditions are important factors accounting for this unusual situation.

The historical roots of the town meeting concept with small semiautonomous health departments hardly provides the basis for comprehensive health services. With few exceptions, attempts at regionalization have not been particularly successful.

Much of the same situation exists in the structure of the local Visiting Nurses' Associations. Small agencies mean small budgets with low salary scale. It is necessary that board members, including staff nurses, become familiar with recent concepts for the care of the chronically ill and aging. This includes the utilization of important therapeutic services such as physical therapy, occupational therapy and social work. The organization and development of home health services is one of the most imposing problems facing the New England States. Other parts of the country are in a more favorable position to develop these services.

The organization and recruiting of the necessary staff to provide the basic home health services will require the cooperation of all professional health and welfare organizations. A minimum of $75,000 in formula grant funds is available from the Public Health Service to assist each State to develop Home Health Services. Some of the initial considerations of the Health Department will include:

1. Identification of deficiency areas.
2. Identification of those areas where there are existing agencies so that they may be assisted to qualify as certified agencies.
3. Encourage development of agencies in areas which have no existing services.
4. Be prepared to provide support through supervision and consultation to those agencies which are already able to qualify.
5. Whenever possible, to expand and improve the good agencies.
6. Develop on a statewide basis opportunities for training courses, seminars and workshops.

The State Health Department will have the responsibility of certifying the vendors or providers of community services. As previously defined, a Home Health Agency must provide skilled nursing and one other therapeutic modality. The agency to be certified also must have written policies developed by a professional group, must make provision for exchange of information, adequate record keeping and maintenance of a clinical record for each patient. Adequate fiscal records must be maintained and the service provided at reasonable cost. As with the current Visiting Nurse Association operations, each patient must have a case plan for treatment outlined and developed by a physician. Among the benefits excluded, would be food services, such as meals on wheels and homemakers per se. This does not exclude our current home health aides who perform personal services.

I feel confident that the basic services can and will be organized which will cover from 40%-60% of the population in New England. As might be expected, the more rural areas and States pose the major problem. (Among other things, the Health Insurance Program provides an opportunity to explore ways of delivering new services that will have an impact on the total medical care pattern in the country.) The responsible State officials will have to work on a priority basis with existing agencies so that maximum coverage is provided by July, 1966. The status of these agency resources in order of increasing complexity include:

1. Local community with existing bedside nursing services.
2. Community with bedside nursing with another agency in the community providing the other modality of therapy.

*This is the second and final part of a paper presented by Dr. Allen at the Harvard School of Public Health January 10, 1966. Part I was published in the May issue of The Journal.

**Regional Program Director for Chronic Diseases, Public Health Service, U.S. Dept. of Health, Education and Welfare, Boston Regional Office, Region I, 120 Boylston Street, Boston, Massachusetts.

3. Traditional hospital-oriented nursing services only.
4. Community having an agency with a therapeutic service but no nursing service; a Medical Social Service Agency is a possibility.
5. And last, but most difficult of all, is trying to establish a service in a community with no nursing service and no other services provided.

The Home Health Service program reemphasizes and brings to the forefront the need for additional, fully trained professional staffs at the State and local level. Immediate attention should be given the strengthening of Public Health Nursing Services for communities throughout the State. *Consideration should also be given for regionalization of political subdivision into functioning medical service areas.*

Caring for the chronically ill and aging is a total community problem that calls for the cooperation and involvement of a multiplicity of agencies and programs. The Home Health Service aspects of the Federal Health Insurance program provides a unique opportunity to explore ways of delivering new services that will have an impact on the total medical care pattern. The end result will most assuredly be an expansion and improvement of community facilities and services that will upgrade the total health care.

Extended Care Facilities

Fortunately, the provision for post-hospital extended care benefits does not go into effect until January 1, 1967. This will provide additional time for the certification process. It is estimated that *not over 20%* of the existing facilities will initially qualify.

To qualify, the facilities must have a transfer agreement with one or more hospitals and must also:

1. Provide skilled nursing care and related services or rehabilitation services to inpatients.
2. Have policies developed with the advice of and reviewed periodically by a professional group, including at least one physician and at least one registered nurse.
3. Have a staff physician, registered nurse, or medical staff responsible for executing such policies.
4. *Have every patient's health care under supervision of a physician and have a physician available for emergencies.*
5. Maintain clinical records on all patients.
6. Provide twenty-four-hour nursing service and have at least one full-time R.N.
7. Provide appropriate methods and procedures for dispensing and administering drugs and biologics.
8. Have a utilization review plan in effect.
9. Be licensed or meet standards for licensing under State or local law, and
10. Meet such other conditions relating to health and safety of patients or to physical facilities, as the Secretary of the Department of Health, Education and Welfare may find necessary.

The Federal Insurance Program will sharpen the focus on the exact picture of the modern nursing home as a place of "dignity, understanding and care for the terminal patient, the massive hemiplegic, for the very old, the debilitated and senile as well as for those with less severe maladies." It will bring the extended care facility in the mainstream of medical care and provide a bridge between the hospital and the home.

The formal relationship provision will provide potential advantages of both the hospital and the care facility. Not least among these will be the exchange of information, joint medical rounds and the extension of inservice training programs to these facilities.

The demand for increasing standards and provision of a complete range of care including PT, OT, and psychiatric consultation have far reaching implications. More hospitals will consider building extensive care facilities and for economy and efficiency of operations the size will gradually increase. Poor economic return will gradually reduce many of the marginal homes. The States must continue a long-range consultation and educational program to upgrade some of the facilities that fall short of the health and safety requirements.

An unresolved issue is the possibility of having separate standards and separate facilities for Medicare recipients and Public Medical Care Beneficiaries. Some States are instituting single standards for both programs to insure adequate quality for all persons needing it.

Let us now turn to Title 19 of the Social Security Amendments which pulls together, under a single formula, the various medical assistance programs. The widespread interest in the Health Insurance Program has overshadowed this aspect of the Medicare Bill which may, in the long run, have a greater impact on health practices and the delivery of care services in this country.

The details of Title 19, including its administration, are still in the formative stages. The three major parts are:

1. Expanded public medical assistance under Kerr-Mills.
2. Expanded child health and welfare programs.
3. Increased payments to existing recipients, including both Social Security and Welfare.

In administering the program at the Federal level, it is generally accepted that the Welfare Administration will be primarily responsible for overall policy formulation and operational management; while the Public Health Service will provide professional consultation. The operation at the State level poses many unresolved questions since, in the majority of States, Welfare has been designated as the sole administrative authority by the Governors. There appears to be a trend toward the development of collaborative agreements with the health authorities for standard setting and arriving at a reasonable cost formula.

It is very much apparent that a tremendous amount of coordination must be done. This is particularly true since:

a. We'fare has broad experience in establishing payment mechanisms;
b. Health Departments have some competence in standard setting;
c. Neither has the necessary competence in setting reasonable costs.

Fortunately, Title 19 has a longer fuse than Title 18. It goes into effect as of January 1 of this year, but permits the States to continue under their existing plans. After December 31, 1969, the Federal Government will discontinue matching for medical care vendor payments through the individual Public Assistance titles.

Of major significance are the inclusion of the concepts of *"equality of medical care"* and comparability of eligibility standards. Title 19 stipulates that all persons aided are to be offered help with the costs in the same amount, same duration and same scope. This will make

Continued on page 128

Maine Heart Association Notes
116 State Street, Augusta, Maine

The Relationship of Cardiovascular Disease To Hyperglycemia

The high prevalence of vascular disease among diabetics has long been recognized and has become increasingly important since the acute complications of diabetes, acidosis, and infection have been controlled.

Since there is strong clinical and laboratory evidence that diabetes mellitus is often an important factor in atherosclerosis, it is surprising that there have been so few studies of carbohydrate metabolism among persons with atherosclerosis. . . . The present report deals with the prevalence of vascular disease among the diabetics encountered in the examination of persons of all ages in a total natural community and with the frequency of hyperglycemia detected among persons in the same population with various manifestations of vascular disease.

. . . The detection of hyperglycemia with significant frequency among persons with each of five different manifestations of vascular disease is a unique finding among epidemiologic studies.

. . . coronary heart disease, other vascular disease (cerebral or peripheral vascular disease), T wave inversion in the electrocardiogram, systolic hypertension, or diastolic hypertension, the proportion with elevated blood glucose levels was significantly greater than among persons of the same sex and age group in the total examined population.

Submitted by Jacob B. Dana, M.D.

Reference—Ostrander, L. D., Jr., et al, Annals of Internal Medicine, Volume 62, Pages 1188-1198, 1965.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Cancer Cells May Have a Secret and Surprising Defense Against Destruction

Research at Harvard Medical School and Massachusetts General Hospital suggests that cancer cells may have a secret and surprising defense against destruction: they may use the body's own basic weapons as a shield.

The American Cancer Society, reporting recently on research by Dr. Kurt J. Bloch, told of cancer's possible perversion of the body's natural resistance to disease.

Antibodies are proteins which the body produces to destroy viruses, bacteria and other disease-causing entities, including tumor cells. Each animal produces several different kinds of antibody proteins called gamma globulins — all aimed at the same invading organism — but each capable of damaging the invader in a different way.

In some animals, such as the guinea pig or mouse, two main types of antibodies are produced: gamma-1 and gamma-2.

When guinea pigs were injected with mouse tumor cells they produced gamma-1 and gamma-2 antibodies against these tumors.

In test tube experiments, isolated gamma-2 antibodies were attached to and clumped tumor cells. With the help of other body defense proteins called "complement", they then ruptured the tumor cells.

Gamma-1 antibodies also were capable of attaching to and clumping tumor cells, but they could not combine with complement and therefore they failed to seriously injure the cancer cells.

Earlier experiments with other model systems had shown that gamma-1 antibodies could protect cells against the destructive action of gamma-2 antibodies plus complement. Dr. Bloch therefore suggested that gamma-1 antibodies against tumor cells might also protect the cells against the more effective attack of gamma-2 antibodies. He also suggested the possibility (still to be tested) that gamma-1 antibodies might stimulate the growth of cancer cells to which they attach.

American Cancer Society News Service Release

In Memoriam

Aroostook County	
Herrick C. Kimball, M.D.	Fort Fairfield
Cumberland County	
Charles L. Cragin, M.D.	Portland
Stanwood E. Fisher, M.D.	Portland
Howard R. Ives, M.D.	Portland
G. E. C. Logan, M.D.	Portland
Jack Manol, M.D.	Portland
Daniel M. Rowe, M.D.	Scarborough Beach
Philip P. Thompson, Sr., M.D.	South Portland
Hancock County	
Raymond E. Weymouth, M.D.	Bar Harbor
Kennebec County	
Adolphe J. Gingras, M.D.	Augusta
Sabro Tashiro, M.D.	Gardiner
Lincoln-Sagadahoc County	
Arthur A. Nichols, M.D.	Edgecomb
Oxford County	
Henry Pearson, M.D.	Brownfield
Penobscot County	
Arthur N. Lieberman, M.D.	Bangor
Piscataquis County	
Ralph C. Stuart, M.D.	Guilford
Washington County	
Charles M. Armstrong, M.D.	Robbinston

County Society Notes

LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held on April 19, 1966 at The Ledges in Wiscasset, Maine.

The President, Alan W. Zeller, M.D., called the meeting to order. The Secretary-Treasurer discussed the Interim Meeting of the Maine Medical Association House of Delegates and announced the proposed increase in M.M.A. dues.

Chris A. Lutes, M.D. of Portland was the guest speaker. He spoke about current techniques of open heart surgery and presented interesting slides.

GEORGE W. BOSTWICK, M.D., *Secretary*

KENNEBEC

The Kennebec County Medical Association met at the Veterans Administration Center in Togus, Maine on April 21, 1966.

The meeting was called to order by the President, Allan J. Stinchfield, M.D.

John D. Denison, M.D. announced a proposed increase in the M.M.A. dues. He also urged that members attend the annual meeting of the M.M.A. in June.

John Abajian, Jr., M.D., Professor of Anesthesia, University of Vermont Medical School, was the guest speaker. He spoke on the problem of shock as related to myocardial infarction, and discussed several points such as stagnation in the capillary bed, micro-coagulation, the inadvisability of using vasopressors and the use of plasma expanders including Dextran and lactated Ringers solution.

LUCIEN F. VEILLEUX, M.D., *Secretary*

CUMBERLAND

Eighty members and guests attended the meeting of the Cumberland County Medical Society held at Valle's Steak House in Portland, Maine on April 21, 1966.

Following a social hour and dinner, Charles R. Glassmire, M.D., introduced a panel discussion of Medical-Legal Interprofessional Association. Edward G. Asherman, M.D. stated that the AMA and the American Bar Association has adopted the National Interprofessional Code as guides for physicians and attorneys. This code has been studied locally by Dr. Asherman's committee and a corresponding committee from the local bar association. Dr. Asherman emphasized that physicians should provide for lawyers a prompt medical report, that conferences should be held at mutually convenient times and places, that arrangements for physicians' appearance in court can usually be arranged to fit into a doctor's schedule, that fees must be reasonable for the time spent by the physician, and that trial fees are the responsibility of the lawyer whereas treatment fees are the patient's responsibility.

George F. Sager, M.D. stated that 75% of trials involve some medical proof or evaluation. He outlined the proper components of a report to a lawyer: circumstances of first contact with patient, patient's version of circumstances, review of symp-

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toms, review of objective findings by physician, diagnostic tests, diagnosis, treatment, prognosis, and a bill for services. Robinson L. Bidwell, M.D. described the mechanics of court procedure, emphasizing that verbal statements are considered legal evidence whereas in medicine we think of evidence as being objective material. He made a parallel of the rigid code of ethics under which both professions work and he defined and described the details of direct examination and cross examination. James H. Bonney, M.D. commented as a member of both professions.

Dr. Morrison announced that the Executive Committee, after investigation, had sent a letter to the UCS supporting the concept of an Information and Referral Center for Chronic Illness. Dr. Glassmire summarized the Interim Meeting of the House of Delegates of the Maine Medical Association and recommended further discussion await the May meeting of the county society. A letter regarding political matters from Lloyd G. Davies, M.D. was received but was tabled until the May meeting, hoping that Dr. Davies would discuss this in person.

Harold N. Burnham, M.D. proposed the formation of a special committee to attempt to anticipate problems referable to Medicare. Following considerable discussion and the recognition that the county society has membership on the Medical Advisory Committee to the Department of Health & Welfare and on the Health Insurance Committee of the Maine Medical Association, the proposal for the formation of a special committee was voted upon and defeated.

STANLEY B. SYLVESTER, M.D., *Secretary*

Deceased

LINCOLN-SAGADAHOC

Arthur A. Nichols, M.D., Edgecomb, Maine, May 14, 1966

News and Notes

100th Anniversary of the Veterans Administration Center, Togus, Maine

Nineteen hundred and sixty-six commemorates the 100th anniversary of the Veterans Administration Center in Togus, Maine. Sunday, August 7, 1966 will mark the opening of the official centennial week. Thursday, August 11, 1966 has been designated as Physician's Day. On this day the practicing physicians in the State of Maine are invited to come to Togus to visit the General Medical, Surgical and Neuropsychiatric facilities. Details of the scientific and social program for Physician's Day will be mailed to each individual physician.

Public Health Service's Health Examination Survey to Visit Cumberland County

The Public Health Service's Health Examination Survey, authorized by Congress in 1956, will visit the Cumberland County area to examine a sample of the teen-aged population (ages 12 through 17 years) during a 4-week period beginning June 29. This will be part of a series of examinations of a nationwide health survey of youngsters.

About 200 youths to be examined in the Cumberland County area will be chosen by a scientific sampling process from the entire 12-through-17-year population. Examinations will be given in the Health Survey's mobile examination center, which will be brought to the area in June and set up in a convenient location. Purpose of the examinations is to collect on a uniform basis statistical information on various aspects of the health of 12-through-17-year-olds and to obtain data on cer-

tain physical and physiological measurements relating to growth and development in this age group.

The process will include a special examination by a physician of the eyes, ears, nose and throat, heart, and neuromuscular systems; a dental examination by a dentist; a test of visual acuity and color vision; recordings of blood pressure; biological and biochemical tests of a blood sample; an audiometric test for hearing performed in a soundproofed room; an X-ray of the hand and wrist for bone age; an X-ray of the chest for cardiovascular and pulmonary abnormalities; a 10-lead electrocardiogram and a phonocardiogram of the heart; a timed vital capacity using a wedge spirometer; an exercise tolerance test under a measured workload using a treadmill; a grip-strength test; recordings of height, weight and various other measurements of growth and development; and measurements of verbal, perceptual and social skills by a psychologist.

The Health Examination Survey is designed to collect data primarily on the health aspects of growth and development. The survey is not intended as a screening procedure; referral for diagnosis is not made. The fact that the examination is not complete and is not a substitute for a visit to one's own physician and dentist is stressed with the parents of each youth examined. No part of the results of the examination except for a card stating the blood type will be disclosed to the youth or his parents. A report of relevant findings will be sent to the youngster's physician and dentist when the parents request that this be done.

The examining physicians will be senior residents or fellows in pediatrics working temporarily with the Public Health Service. Other members of the examining team will include a nurse, a dentist, two psychologists, and X-ray and other technicians regularly on the PHS staff.

This health examination of youth is referred to as the 3rd Cycle of the Health Examination Survey. Earlier, a 2nd Cycle of children's (ages 6 through 11 years) health examinations was completed last December. Over a 30-month period, a total of 7,129 children were examined in 40 locations across the Nation. A 1st Cycle of adult health examinations ending in late 1962 completed a 3-year survey and examined a total of 6,672 adults (ages 18 through 79 years).

Book Reviews

Gonadotropins: Physicochemical and Immunological Properties, Edited by G. E. W. Wolstenholme and Julie Knight. Published by Little, Brown and Company, Boston, 1965. Price $3.50.

This small book is a transcript of the one-day symposium on gonadotropins sponsored by Ciba Foundation. There were eleven contributors including C. A. Gemzell. The presentation is concerned mainly with the purification of FSH, LH, and chorionic gonadotropin and identification of these compounds by immunological and biological means. For anyone interested in the latest methods used to purify these compounds the book has a wealth of detail and there is some discussion of what the future holds for those of us who may make use of these hormones and techniques.

STANLEY W. KENT, M.D., Portland, Maine

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Applied Hypnosis and Positive Suggestion, Edited by George A. Ulett and Donald B. Peterson. Published by C. V. Mosby, St. Louis, 1965. Price $8.50.

This is a very good concise text dealing with the basics of hypnosis as applied to the healing arts.

Without being encyclopedic, it is written for the busy practitioner who already has a great deal to read. With it one can get a beginning knowledge of the uses and techniques.

CLARENCE E. MCINTIRE, D.M.D., Portland, Maine

Letter to the Editor

To the Editor:

After struggling to make the May 31st deadline to set up our hospital's Utilization Committee, you can understand how frustrated I felt by the following story. A patient of mine who recently sold a prosperous business and who had Blue Cross-Blue Shield coverage recently entered the Togus VA Hospital for a routine herniorrhaphy. His wife told me that the operation was performed two weeks after he entered the hospital. I asked her why they kept him there so long, and she said, "Well, they examined him, and then they sent him home on a weekend pass. He felt they were wasting his time."

I have had a chance to speak to several physicians on the staff at Togus since then, and none has even heard of a Utilization Committee!

It is well known amongst doctors practicing at Veterans Administration Hospitals that the money the hospital receives from the Government is directly proportional to the average daily patient load. If the hospital stays filled the appropriation remains more or less constant. This has a demoralizing effect upon the doctors who work there because obviously they realize they can never get caught up – the minute they discharge a patient another is admitted. It also has a deleterious effect upon the veteran. The tendency to keep the patient hospitalized as long as possible keeps other veterans from being admitted. The last time I tried to admit a patient I was told there were 158 cases ahead of him on the waiting list.

How long is the medical profession going to tolerate this double standard? When are they going to insist that Veterans Administration Hospitals also have Utilization Committees? After all, what's sauce for the goose is sauce for the gander.

LOUIS BACHRACH, M.D., Brunswick, Maine

Department of Health and Welfare

Continued from page 122

possible the realization of one, single level of medical care standards for the entire population.

One other implication is concerned with professional manpower. Regulations state that the approved State Plan for the Title 19 Program must include a separate and distinct unit within the administrative agency. It further specifies that the unit be headed by a medical doctor with public health training or a medical social worker with public health training. The schools of public health have a real responsibility to expand existing programs to train the required competent leadership.

In summary, Dr. Ellen Winston, Commissioner of Welfare, has stated that the welfare provisions of this landmark legislation will go a long way towards assuring that no American would be deprived of the health or medical care he needs because he is too poor to pay the

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Meprobamate: Drowsiness may occur and can be associated with ataxia, the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor, and respiratory collapse.



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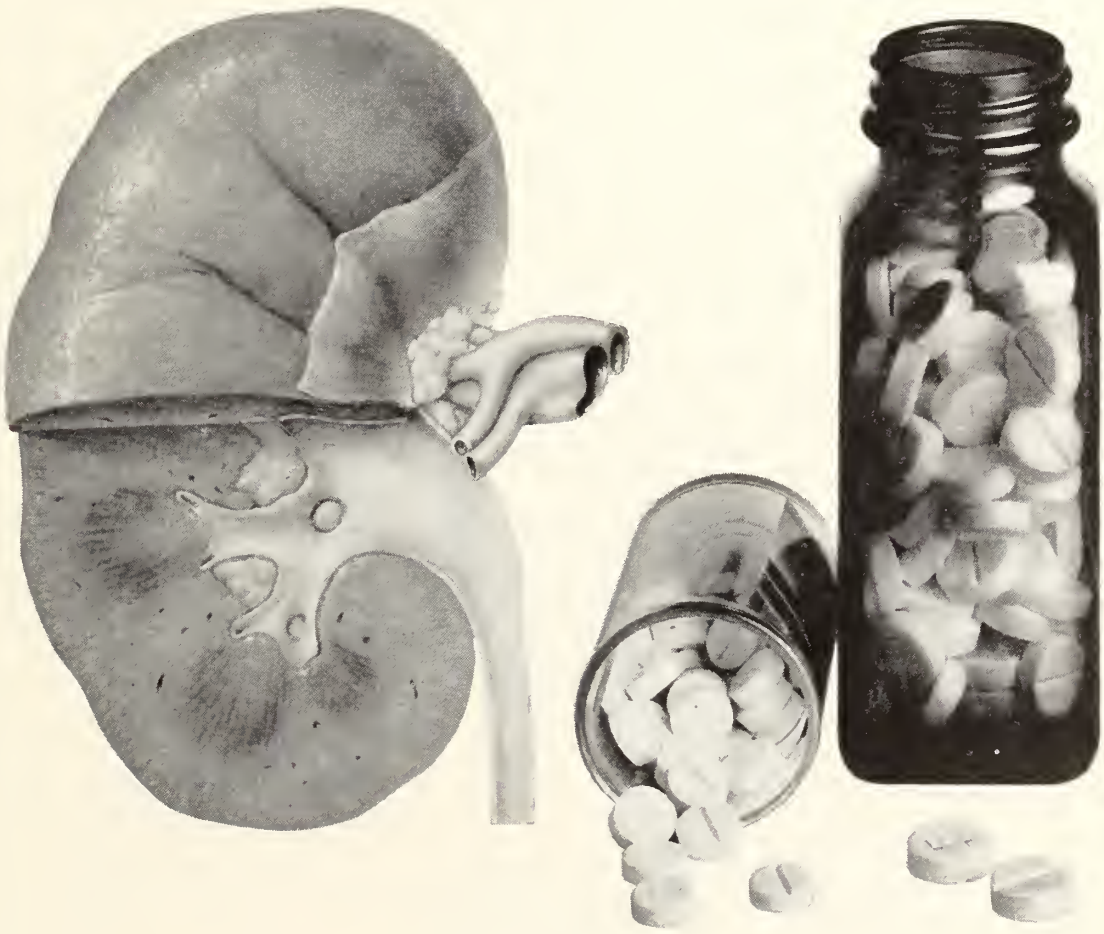
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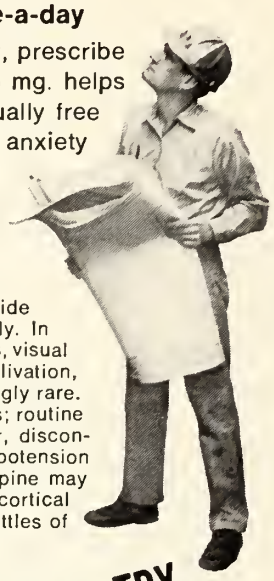
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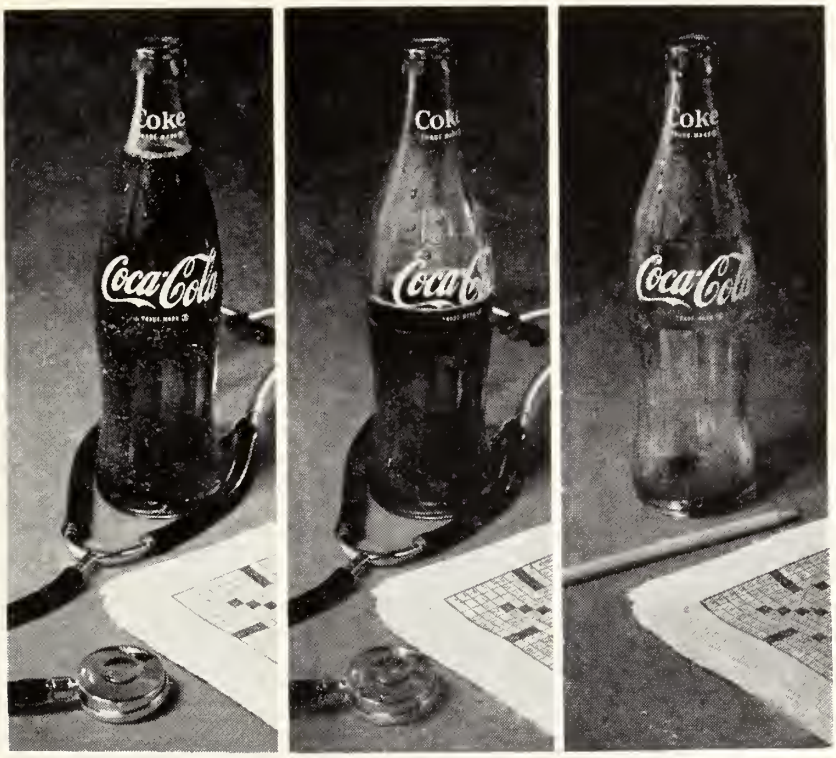
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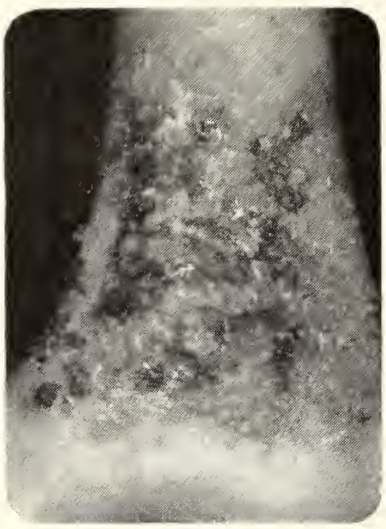
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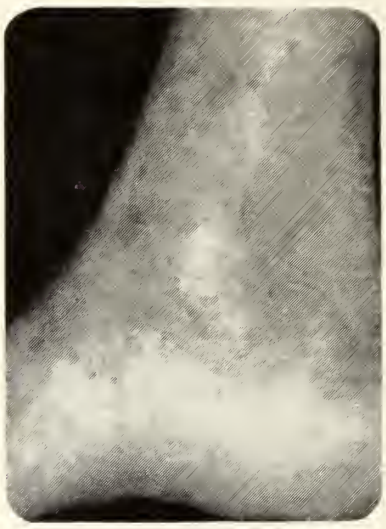
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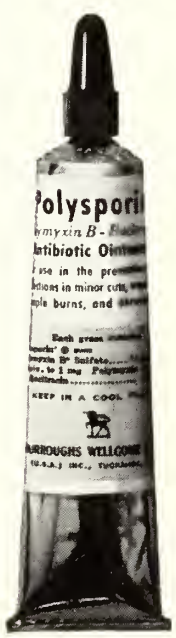
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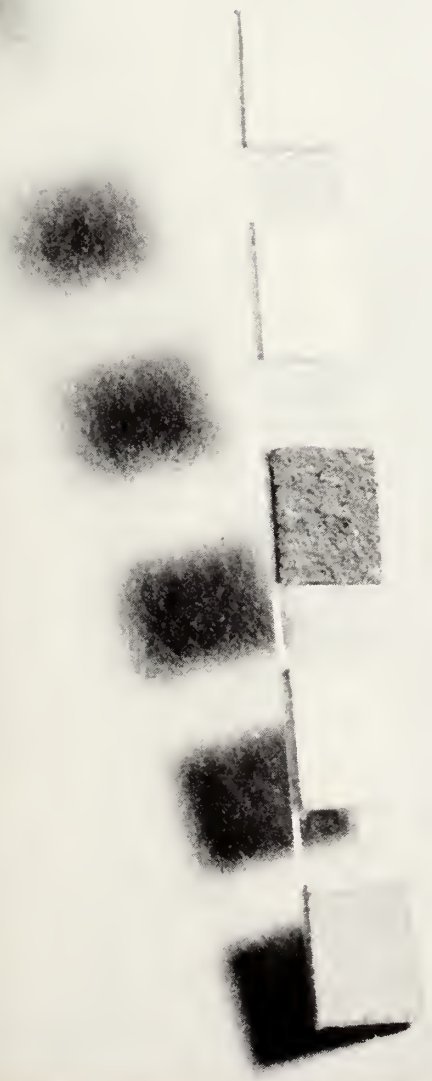
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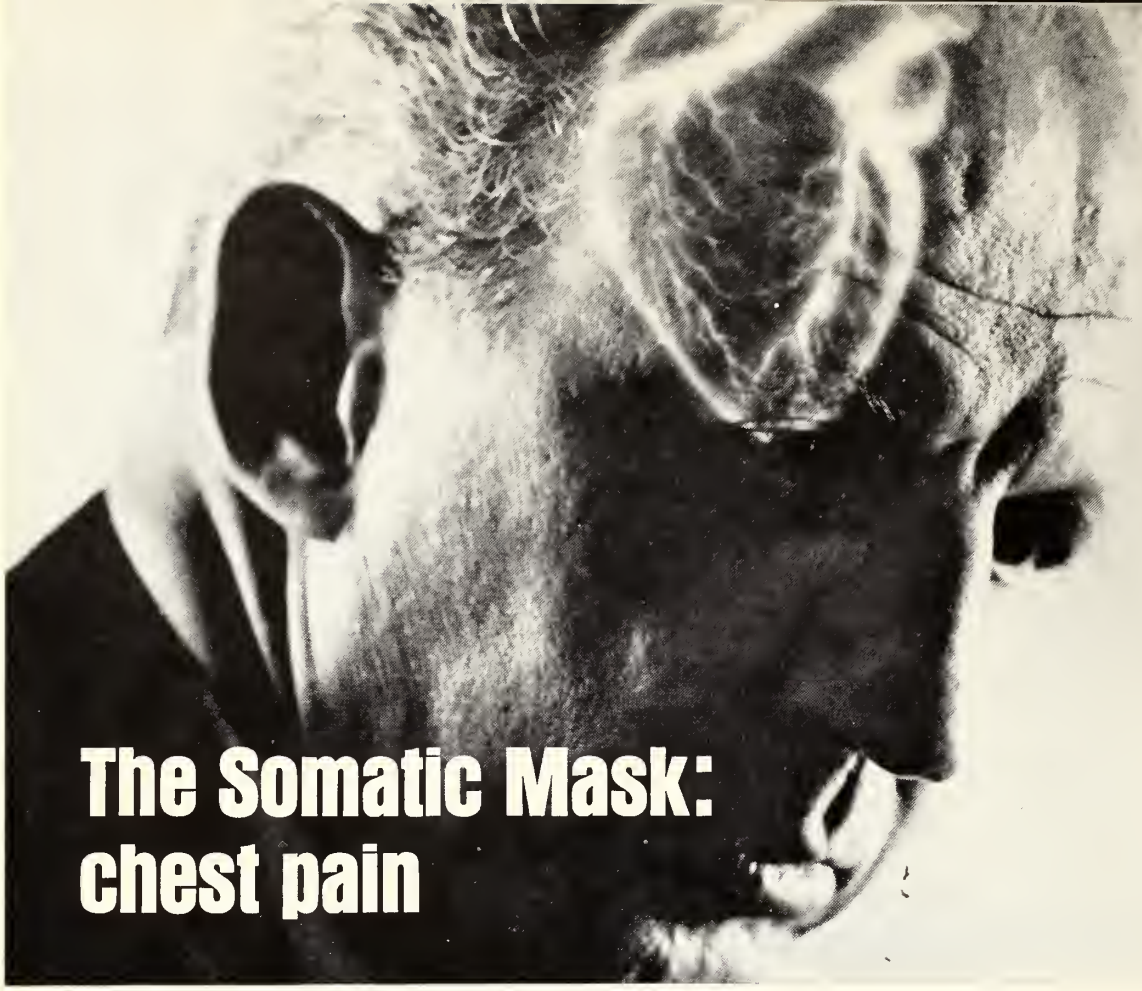
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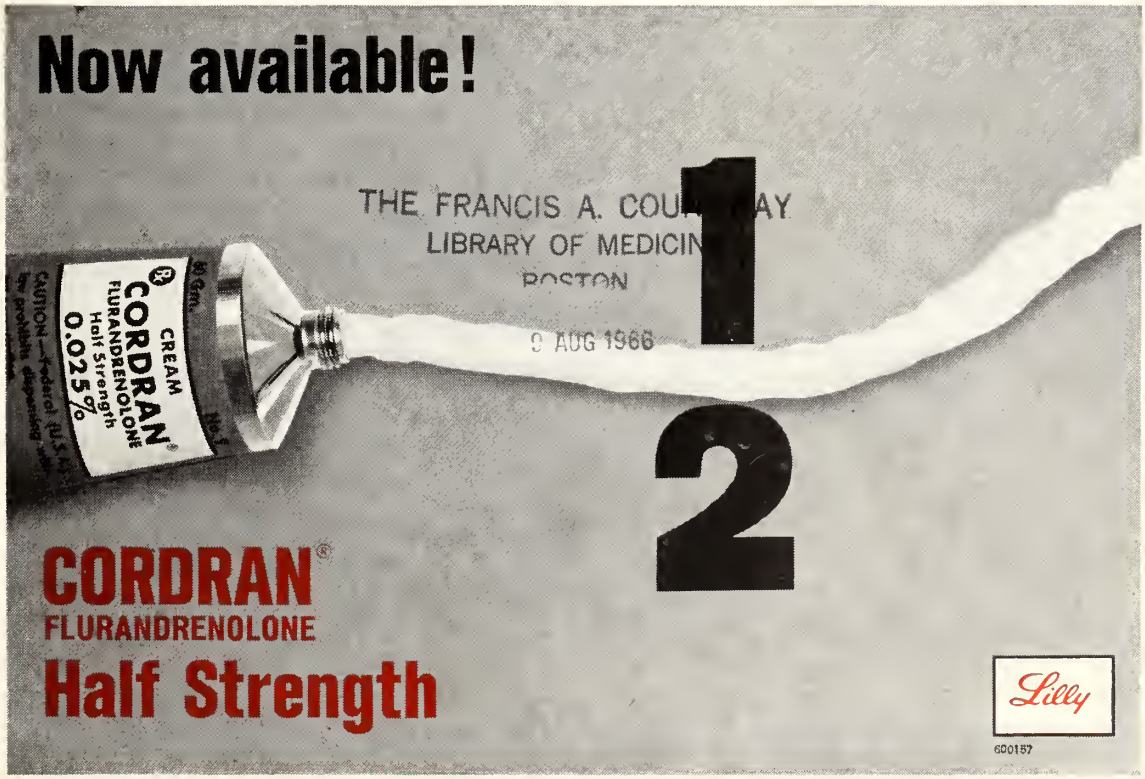
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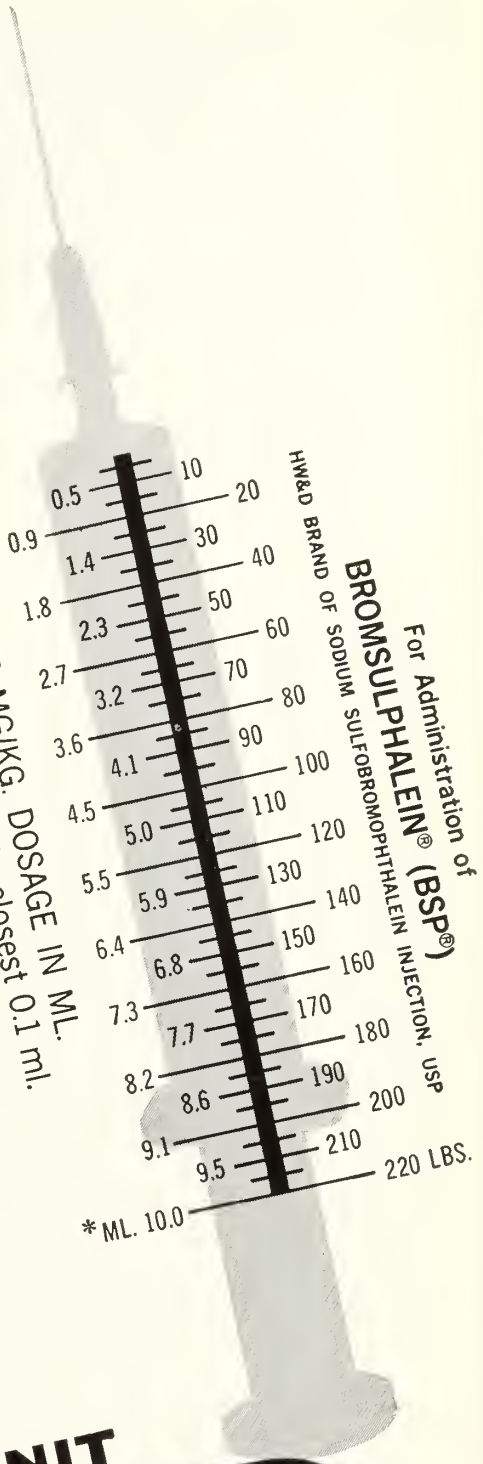
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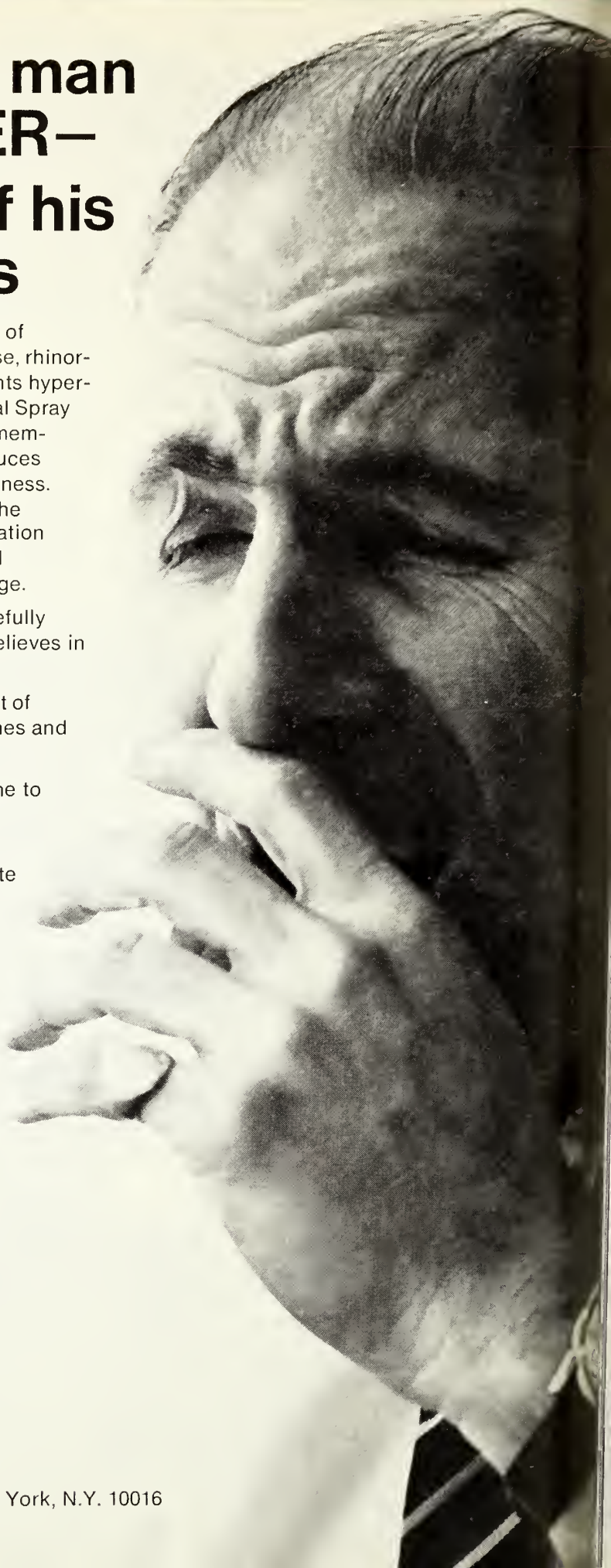
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Non-Specificity Versus Specificity in Alcoholism*

C. Rothstein, Ph.D., I. Zeltzerman, M.D. and P. Siegel, M.A., M.S.

Introduction

The abuse of alcohol, or at least the recognition of this, has increased markedly in the past few years.[1] The proliferation of local, state, national and even international alcohol research and rehabilitation centers attests to the growing concern with this problem. As of 1962, the Classified Abstract Archive of the alcohol literature reported over eight thousand articles dealing with this topic.[2] Keller estimates that between four and five million Americans can be considered to be alcoholics.[3] Adding to this figure another ten to fifteen million dependents of alcoholics, the number of people adversely affected by alcohol abuse mushrooms to between 14 and 20 million. In the past year approximately 43% of psychiatric admissions to this hospital had a primary or secondary diagnosis of alcoholism.

Paralleling the marked increase in concern with alcoholism, have been the speculations as to its etiology. The etiological hypotheses advanced center mainly in the areas of psychological sociocultural, and physiological factors – or admixtures of these.[4,5] Discernible among the psychological speculations are two contrasting points of view[6] which may be labeled specificity and non-specificity theories. The specificity theorists tend to view alcoholism as arising in a population with a set of problems and/or a personality configuration unique to this group. The less numerous non-specificity theorists tend to view alcoholism as a symptom arising in a population with problems and/or a personality configuration no different from that to be found in non-alcoholic emotionally disturbed populations. The authors share this latter view. In essence, we see alcoholism as a symptom (whose aim, as with any symptom, is to avoid the painful affects associated with an inability to solve life problems) which arises in a population essentially similar, from a personality standpoint, to other psychiatric populations. And while it is quite likely that sociocultural and/or physical factors may influence the choice of this mode of defense (and may differentially affect the secondary problems exacerbated by this defense) we posit that no basic personality differences will be found between alcoholic and non-alcoholic psychiatric populations.

Present Study

Method and Subjects: As part of a general research program at the Togus Veterans Administration Neuropsychiatric Research Laboratory, all psychiatric patients admitted to this hospital between June, 1963 and June, 1964 were rated on certain demographic, historical, social and psychiatric variables. The scores were then coded on Royal McBee coding cards which provided us with a central source of data for all psychiatric patients admitted to this hospital during that period. Patients' admission diagnoses were also affixed to each card.

This intake population was divided into four groups matched for age and education. Group A was composed of patients with a primary diagnosis of alcoholism, the secondary diagnosis, where present, ranging among the neurotic and character disorder classifications (N=88). Group B comprised patients with a secondary diagnosis of alcoholism, the primary diagnosis ranging among the neurotic and character disorder classifications (N=94). Group C was composed of individuals with a primary diagnosis among the neurotic, character disorder, or affective classifications, no alcohol symptom being present in this population's diagnosis (N=115). Group D consisted of patients with a primary diagnosis of schizophrenia, no alcohol being present in this population (N=79). Patients with a primary chronic brain syndrome diagnosis were deleted from this study population. The average age of the total study population was 44 years, and the average educational level was 10.2 years.

Central to this study is the analysis of the similarities and differences among alcoholic and non-alcoholic psy-

*From the Veterans Administration Center, Togus, Maine.

TABLE 1

BACKGROUND DATA

Variables	*Primary Alcoholic (N=88)*	*Secondary Alcoholic (N=94)*	*Mixed Diagnostic (N=115)*	*Schizophrenic (N=79)*
Broken Homes	28%	25%	26%	21%
Rural Background	36%	26%	28%	37%
Father's Occupation				
1. Professional-Managerial	13.6%	19.6%	14.3%	18.9%
2 Clerical-Sales	3.4%	6.5%	8.9%	8.1%
3. Service	5.7%	6.5%	12.5%	6.8%
4. Agricultural-Fishing	14.8%	9.8%	17.9%	14.9%
5. Skilled	26.1%	30.4%	24.1%	18.9%
6. Semi-skilled	20.5%	15.2%	7.1%	16.2%
7. Unskilled	12.5%	12.0%	15.2%	16.2%
Patient's Religion				
1. Protestant	56%	53%	67%	49%
2. Catholic	44%	47%	33%	51%
Age at Separation from Parental Home	M=23.3 s.d.=9.1	M=20.9 s.d.=5.5	M=23.1 s.d.=9.3	M=26.1 s.d.=9.8

chiatric populations on those variables comprising our intake form.

Results: Table 1 sums the comparisons on the background variables.* The percentages from broken homes and from rural backgrounds do not differentiate the populations. Likewise, distribution of father's occupation is similar for all groups, as is religion. The one area of difference on background variables lies in age at separation from parental home. The primary alcoholic group tends to separate from the parental home at a significantly younger age than the schizophrenics, at a significantly older age than secondary alcoholics, and at the same age as the mixed diagnostic group.

Table 2 presents the comparisons on the social adjustment variables. The distribution of patients' occupational level is similar among the groups. However, the primary alcoholic group shows a work history significantly more stable than the schizophrenic group, significantly less stable than that of the mixed diagnostic group, and as stable as the secondary alcoholic group. On the variable "never married," the primary alcoholic group has a significantly lower percentage than the schizophrenic group but cannot be differentiated from the other two populations. The primary alcoholic group has a significantly higher divorce rate than the mixed diagnostic group, but is not significantly different from the other groups. Length of military service does not differentiate the primary alcoholic group from the other populations, nor does "percent in combat."

Table 3 presents the variables dealing with hospital history. The primary alcoholic group tends to have their first psychiatric hospitalization occur at a significantly later point in life than the secondary alcoholic and

*In all tables Chi-square analysis was employed for those figures expressed as percentages; T tests were utilized for those figures expressed as means.

TABLE 2

SOCIAL ADJUSTMENT DATA

Variables	*Primary Alcoholic (N=88)*	*Secondary Alcoholic (N=94)*	*Mixed Diagnostic (N=115)*	*Schizophrenic (N=79)*
Patient's Occupation				
1. Professional-Managerial	13.6%	7.4%	8.8%	7.6%
2. Clerical-Sales	7.9%	16.0%	10.5%	6.3%
3. Service	7.9%	4.3%	4.4%	6.3%
4. Agricultural-Fishing	1.1%	6.4%	5.3%	8.9%
5. Skilled	19.3%	14.9%	14.9%	17.7%
6. Semi-skilled	25.0%	19.1%	21.9%	13.9%
7. Unskilled	21.6%	29.8%	28.1%	25.3%
8. Other	3.4%	2.1%	6.1%	13.9%
Unstable Work History	47%	38%	30%	67%
Never Married Rate	15%	7%	9%	34%
Divorce Rate	36%	40%	20%	27%
Length of Military Service (Months)	M=35.1 s.d.=16.8	M=37.7 s.d.=16.3	M=37.3 s.d.=19.8	M=32.9 s.d.=15.3
Percent in Combat	36%	27%	29%	29%

schizophrenic groups. The total number of psychiatric hospitalizations, total time in psychiatric hospitals, and longest single psychiatric hospitalization significantly differentiates the primary alcoholic group from the schizophrenic group but not from the other groups. (All three groups were significantly lower on these variables than the schizophrenic group.)

The primary alcoholic group had significantly more general medical hospitalizations than any of the other populations, but the incidence of psychosomatic disorders did not differentiate the primary alcoholic group from the other populations. The percentage of primary alcoholics having a history of extensive use of psychotrophic drugs is significantly less than for the schizophrenic population but is not significantly different from the other two populations.

Table 4 sums the comparisons on the psychiatric symptoms. These are expressed in terms of percentage of patients in each group rated as manifesting the symptoms. On the symptoms "anxiety," "depression" and "tension," the primary alcoholic group scored significantly higher than the schizophrenic group, but did not differ significantly from the other two groups. Guilt feelings occur with a significantly greater frequency in the primary alcohol group than in both the mixed and schizophrenic populations. "Somatic concern" was significantly less frequently found in primary alcoholics when compared to the mixed diagnostic groups. Hostility did not significantly differentiate any of the groups. "Suspiciousness" and "uncooperativeness" were significantly higher in the schizophrenic group than in the three remaining groups. On the symptom "emotional withdrawal," the primary alcoholic group was significantly lower than both the schizophrenic and mixed diagnostic

TABLE 3

HOSPITAL HISTORY

Variables	*Primary Alcoholic (N=88)*	*Secondary Alcoholic (N=94)*	*Mixed Diagnostic (N=115)*	*Schizophrenic (N=79)*
Age at First Psychiatric Hospitalization	M=39.3 s.d.=9.7	M=36.3 s.d.=9.0	M=37.7 s.d.=10.9	M=33.5 s.d.=9.9
Total Number of Psychiatric Hospitalizations	M=2.7 s.d.=3.2	M=2.8 s.d.=2.6	M=2.4 s.d.=1.7	M=4.2 s.d.=3.6
Total Time in Psychiatric Hospitals (months)	M=4.6 s.d.=7.0	M=6.0 s.d.=9.7	M=5.6 s.d.=8.4	M=30.8 s.d.=41.3
Longest Single Psychiatric Hospitalization (months)	M=3.0 s.d.=3.8	M=3.3 s.d.=4.5	M=3.3 s.d.=3.9	M=21.5 s.d.=36.7
Number of Admissions to General Medical Hospitals	M=3.3 s.d.=3.6	M=2.3 s.d.=2.4	M=2.5 s.d.=3.2	M=1.5 s.d.=1.7
Incidence of Psychosomatic Disorder	47%	39%	57%	56%
Heavy Use of Psychotropic Drugs	19%	29%	30%	43%

TABLE 4

PSYCHIATRIC SYMPTOMS

Variables	*Primary Alcoholic (N=88)*	*Secondary Alcoholic (N=94)*	*Mixed Diagnostic (N=115)*	*Schizophrenic (N=79)*
Anxiety	80%	74%	75%	54%
Depression	68%	76%	73%	52%
Tension	77%	70%	68%	51%
Guilt Feelings	70%	61%	52%	46%
Somatic Concern	42%	40%	64%	38%
Hostility	14%	19%	11%	20%
Suspiciousness	16%	19%	19%	42%
Uncooperativeness	2%	3%	6%	14%
Emotional Withdrawal	12%	21%	28%	57%
Blunted Affect	8%	19%	21%	48%
Motor Retardation	23%	12%	12%	24%
Mannerisms and Posturing	5%	2%	5%	16%
Grandiosity	12%	15%	18%	33%
Unusual Thought Content	8%	7%	10%	33%
Conceptual Disorganization	8%	11%	14%	39%
Hallucinatory Behavior	19%	11%	13%	32%

groups. On "blunted affect" the primary alcoholic group was significantly lower than the other three. "Motor retardation" significantly differentiated the primary alcoholic group from the secondary alcoholic group and mixed diagnostic groups, but not from the schizophrenic group. On the symptoms "mannerisms and posturing," "grandiosity," "unusual thought content," "conceptual disorganization," and "hallucinatory behavior," the primary alcoholic group, secondary alcoholic group, and mixed diagnostic groups were all significantly lower than the schizophrenic group while equal to each other.

DISCUSSION

While the majority of background and social adjustment variables do not differentiate the study populations, three key variables – "age at separation from parental home," "percent never married" and "work instability" do significantly differentiate the primary alcoholic secondary alcoholic and mixed diagnostic groups from the schizophrenic group, but not from each other. These findings suggest that the former three groups contain individuals with a level of ego-integration quite similar to each other while somewhat higher than that attained by individuals in the schizophrenic group.

The data on the hospital history variables provides corroborative evidence for the above interpretation. The observation that the primary alcoholic, secondary alcoholic and mixed diagnostic groups have a psychiatric hospital history pattern quite similar to each other but quite different from the schizophrenic group, is again in the direction of the inference that these three groups have a level of ego-integration equally superior to that achieved by the schizophrenic group.

The symptom variables provide data consistent with the other findings. By and large, the majority of the symptoms do not differentiate the primary alcoholic, secondary alcoholic and mixed diagnostic groups from each other, but do significantly differentiate these groups from the schizophrenic group. The former three populations compared to the schizophrenic group show a significantly higher incidence of those symptoms associated with uncompensated affect and less severe levels of ego-fragmentation, i.e., anxiety, depression, tension and guilt, whereas the schizophrenic group shows a significantly greater incidence of those symptoms associated with more severe ego-disintegration, i.e., suspiciousness, emotional withdrawal, blunted affect, mannerisms and posturing, grandiosity, unusual thought content, conceptual disorganization and hallucinatory behavior. While, of course, the greater incidence of these severely pathological symptoms is expected in the schizophrenic group, inasmuch as many of these symptoms provide cues for the diagnosis of schizophrenia, the symptom picture of the other three groups highlights their similarities to each other and their differences from the schizophrenic group.

Within the scope of the variables employed here a non-specificity view of alcoholism vis-a-vis other non-schizophrenic psychiatric populations seems supported. It appears that there is little that differentiates the non-schizophrenic populations from each other. The only variables showing some differences among the three non-

schizophrenic groups suggest that heavy alcohol intake interferes somewhat with the ability to maintain a job and to stay married, as well as leading to a greater frequency of general medical hospitalizations. On the other hand, heavy alcohol intake appears to serve as a social lubricant as reflected by the primary alcoholic group's attaining lower percentages than the other two non-schizophrenic populations on the variables "emotional withdrawal" and "blunted affect."

All three non-schizophrenic groups appear to be dissimilar to the schizophrenic group on several important variables which cut across background, social adjustment, hospital history and symptom areas. These differences occur with sufficient frequency and are of sufficient magnitude to suggest that the population defined by a schizophrenic diagnosis is in many ways different from the populations defined by the other diagnostic groupings. These differences appear to be in the direction of a similarly* higher level of ego-integration in the non-schizophrenic populations vis-a-vis the schizophrenic group. It is likely that the differences between non-schizophrenic and schizophrenic groups would be even greater in a non-veteran population, inasmuch as military service provides a screening process and the proportion of reactive to process schizophrenics is probably greater in the veteran population than in a non-veteran population. Also deserving mention here is the finding that the variance estimates for many of the variables in the schizophrenic sample were large and heterogenous with respect to the other samples, suggesting that the symptom diagnosis "schizophrenia" may be a relatively poor method for grouping individuals.

One suspects that those studies reporting major differences between alcoholic and other populations may have obtained such differences as a function of the nature of the comparison populations.[7,8,9,10] If an alcoholic population is compared to a normal* or schizophrenic population, one would expect to find differences –differences which may wash out when the comparison populations encompass a wide range of psychiatric groups of similar age, education and hospital status.[11] Secondly, those findings delineating alcoholic populations from other populations may reflect differences that follow symptom choice,[12] e.g., the secondary social and physical problems exacerbated by alcohol abuse, rather than reflecting differences inherent in the pre-symptom personality of the individuals.

None of the data of our study provides clues as to those factors relating to symptom choice. While it may be true that sociocultural and/or physical factors affect symptom choice and the secondary problems exacerbated by specific symptoms, there is little in the study to suggest that alcoholism stems from a personality pattern or a range of problems different from those to be found in other non-schizophrenic psychiatric populations. Consistent with this observation is the belief that the treatment of alcoholics, from a personality standpoint, should be essentially similar to the treatment employed with other non-schizophrenic psychiatric populations.

Summary

A specificity versus non-specificity view of alcoholism was evaluated by comparing a primary alcoholic group to secondary alcoholic, mixed diagnostic, and schizophrenic groups on background, social adjustment, hospital history and symptom variables. The data was interpreted as supporting a non-specificity view of alcoholism vis-a-vis other non-schizophrenic psychiatric populations.

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*Consonant with the position that alcoholic populations are not *uniquely* different from normal populations was the finding that all of our psychiatric groups were significantly differentiated from the normal veteran population of Maine on the variables: education, occupational level, and divorce rate. (United States Census of Population 1960, Maine — Detailed Characteristics.)

Alcoholic Rehabilitation at Togus — A Progress Report

EDWARD V. MALCOM, Ph.D.*

The alcoholic unit, organized in 1963 and reported in this Journal in July, 1964 by Dr. Paul Fletcher and Dr. Edward V. Malcom, continues its operation, now under the direction of Dr. Ory D. Canal.

The unit is now in its fourth year, its current title changed to "Abnormal Drinkers Therapy Unit" (ADTU) in order to emphasize its function and focus on the problem. Research on personality types underlying abnormal drinking is still in the process of data gathering The Unit treats the patient suffering from abnormal drinking problems as the emotionally, and often physically, ill person that he is. The emphasis of treatment is group and individual psychotherapy. We have a major interest in the behavioral therapy process which will promote, or "train in" new and effective interests and habit patterns which are needed to produce the feeling of well-being which they had vainly sought through the use of alcohol. The patient is encouraged to face the realities of his life and the resolution of his problems without the use of alcohol or other supportive drugs. An effort is made to help the patient recognize his inability to stop drinking, for reasons beyond his control after his "first drink." Lectures, films, and discussions on the physical, chemical, and physiological aspects of alcoholism are featured in the therapy program. Families of the abnormal drinkers continue to be encouraged to interview members of the team involved in the therapy program.

The team approach is emphasized and each patient of the Unit is seen by the team comprising the Unit's director and ward physician, the psychologist, the social worker, the ward nursing personnel, and a chaplain.

"Follow-through" continues to be a necessary part of the program. Contact with patients includes the use of the telephone, the mail, and personal interviews. Patients are encouraged to keep in touch with members of the team and to visit the hospital at their convenience during working hours when members of the team are present. Experience has shown that effective follow-through is less time-consuming than we had anticipated. Patients who are discharged or placed on trial visit need reassurance, at first frequently, but in rapidly diminishing increments as they become better stabilized in their communities. Patients are encouraged to return to the hospital when they feel tense or in need of hospitalization before slipping too deeply into their old alcoholic patterns.

"Success" – that is, patients who have made marked improvement in either controlling their abnormal drinking pattern or who are now abstaining altogether, remains at approximately the 60% mark established in our last report.

At this point, we have established an effective in-service treatment program and follow-through program. We recognize the pressing need for "half-way house" facilities to provide a link between the institution and the community itself. It is this missing link which provides the adequate housing, controls, and social environment so necessary. The patient who leaves the hospital without a family or community contacts finds it difficult to reestablish himself in the community when there is no resource of this type available, and is apt to revert quickly to his old patterns.

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100th Anniversary of the Veterans Administration Center, Togus, Maine

Nineteen hundred and sixty-six commemorates the 100th anniversary of the Veterans Administration Center in Togus, Maine. Sunday, August 7, 1966 will mark the opening of the official centennial week. Thursday, August 11, 1966 has been designated as Physicians' Day. On this day the practicing physicians in the State of Maine are invited to come to Togus to visit the General Medical, Surgical and Neuropsychiatric facilities. Details of the scientific and social program for Physicians' Day will be mailed to each individual physician.

A Change in Dental Lamina Dura Terms for the Clinician

LAWRENCE PARIS, D.D.S.*

The dental lamina dura is neither a discrete organ nor a specialized tissue. Normally, the bony walls of the tooth sockets in the jaw bones show a cortical-like thickening and are continuous with the cortex of the alveolar bone. On a dental x-ray film, this cortical-like thickening appears as a radiopaque line and is called the lamina dura. The density of this line can vary in diseases which involve the withdrawal of calcium from the bone, i.e. hyperparathyroidism.

To describe the "complete loss" or "partial loss" of the lamina dura in the presence of a complete socket around a tooth root is not correct because a socket wall is present. These walls may be said to be missing only in areas of occasional cystic-like radiolucencies or where there is no tooth root. Better descriptive terms would be "complete loss of definition" of the lamina dura from the adjacent bone, or "partial loss of definition" of the lamina dura from the adjacent bone.

The present descriptive terms infer that the lamina dura interpretation is a positive and quantitative, a yes and no, black and white, present and not present, diagnostic sign. Actually, this interpretation is a qualitative, sliding-scale type of reading which is always more difficult to evaluate than the former and can never be measured exactly. For these reasons of clearer communication and descriptive accuracy, this change in terminology is recommended.

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Quadriplegia in Hyperpotassemia: A Case Report

MEYER EMANUEL, M.D.* and ROGER G. METCALF, M.D.**

Despite the many unknowns and the attainability only of an uncertain degree of precision, the application of the principles of fluid and electrolyte balance is a commonplace of modern hospital practice. Occasionally a severe imbalance calls for prompt corrective measures. One such instance is hyperpotassemia or hyperkalemia. The voluminous literature on electrolytes notes that flaccid paralysis of striate muscle may occur in extreme elevations of the serum potassium.[2,3] Because this manifestation of serum potassium excess, although known to exist, is encountered infrequently we are reporting our experience in one such instance. We wish to call attention to the unheralded onset of the manifestation and the fact that with proper corrective measures it is reversible.

CASE REPORT

A 50-year-old man was admitted to the Veterans Administration Center, Togus, Maine, on Jan. 5, 1966, because of bleeding from a permanent nephrostomy tube in his left solitary kidney. The right kidney previously had been removed for advanced calculus disease in 1956. Subsequently stones formed in the remaining left kidney and these were removed in 1958 only to recur in the presence of chronic infection. In 1961 he was admitted with oliguria, a BUN of 314 mg. %, creatinine of 5.5 mg. %, venous CO_2 of 36 vol. % or 10 mEq./L., and a serum potassium of 8.5 mEq./L. It was necessary to subject him to extracorporeal hemodialysis before an heroic nephrolithotomy could be done to remove only the bulk of the calcific sludge and to establish a permanent nephrostomy as the only practical solution.

In 1962 a second hemodialysis was required when he was admitted with a BUN of 180 mg. % and a serum potassium of 7.9 mEq./L. Further surgery could not be considered in the certain knowledge that this would mean disruption of the friable renal parenchyma encased in dense scar tissue and possible demise from a massive, uncontrollable hemorrhage. He was seen subsequently in numerous follow-up visits and readmissions chiefly to change the nephrostomy tube which was often filled with phosphatic debris, clots, and a mucoid exudate. Difficulties in maintaining a patent nephrostomy included the Pseudomonas and Proteus infection refractory to urinary antiseptics, recurrent bleeding, and a low-level socioeconomic environment. Self-irrigation with antiseptic and Suby solutions at his home proved to be impractical. The usual treatment in the hospital included change of the tube, a course of antibiotics, increased fluid intake and attention to personal hygiene. An occasional transfusion was given. There was usually rapid improvement in the BUN, nourishment, and morale. At no time could permanent removal of the nephrostomy tube be entertained since the constantly forming phosphatic stone sludge frequently blocked the scar-distorted upper ureter.

When he entered Togus on Jan. 5, 1966, he presented the usual picture as described above. The BUN was 70 mg. % and the urine was infected with Proteus. The nephrostomy

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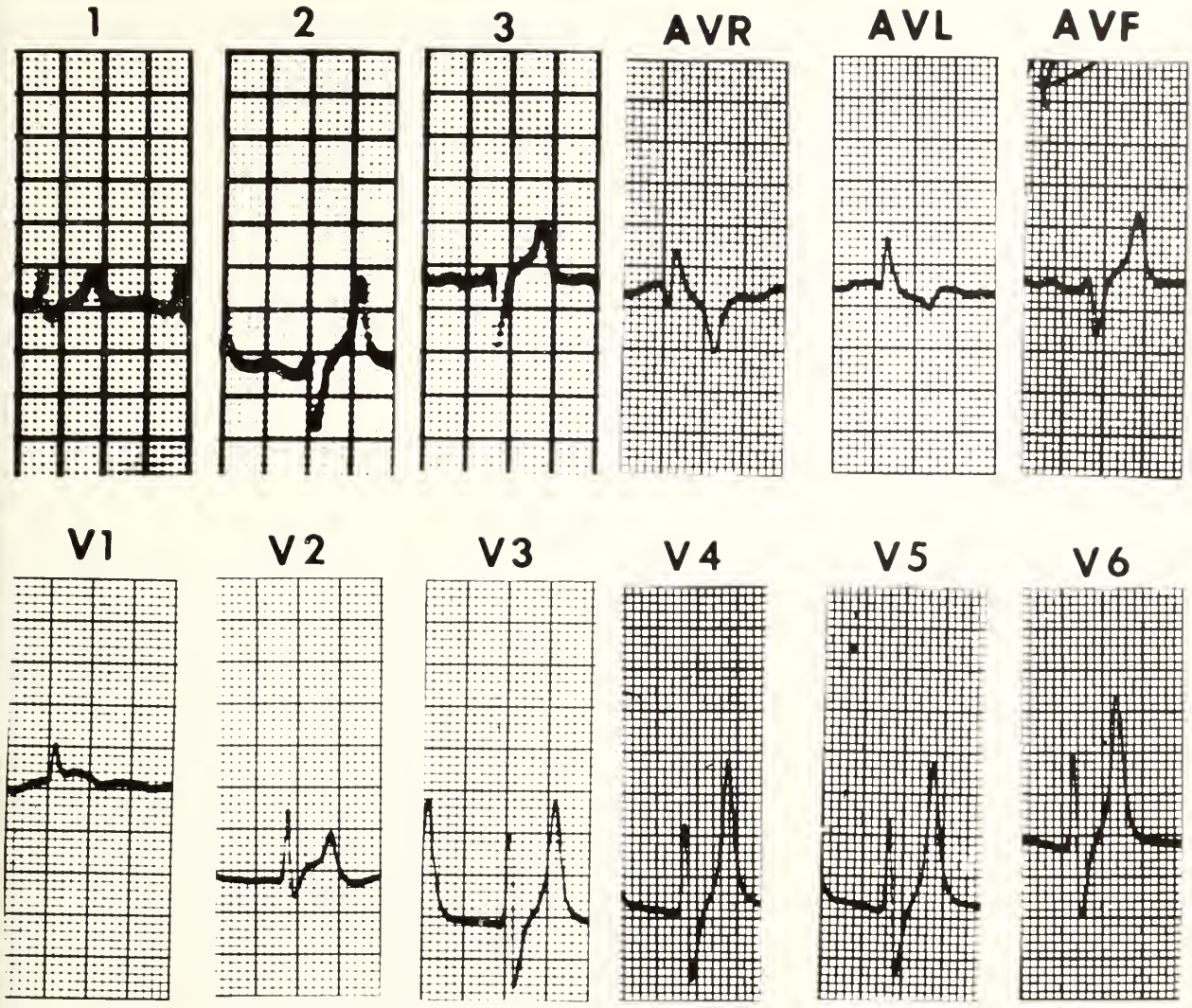


Fig. 1(a). Electrocardiogram taken Jan. 10, 1966, showing tall, peaked T waves and wide QRS complexes characteristic of hyperpotassemia.

tube was draining blood-stained urine but was not obviously blocked. He was started on the treatment regime outlined and improvement, with some complacency, was expected to follow. A mild nausea 2 days later was blamed on the antibiotic capsules. He was walking about comfortably and was not observed too closely.

On Jan. 10 the nephrostomy tube was reported to be blocked by clots of increased bleeding. It was removed and replaced. The mild nausea continued and the patient took to his bed late in the day. At 5:45 p.m. a nurse recorded a blood pressure of 110/70, (admission blood pressure was 110/90) with a pulse of 96. At 7:00 p.m. he was visited and appeared weak, pale, with a cool skin, and in a mild shock-like state with the superficial veins collapsed. Despite a mild anxiety he could best be described as apathetic. Casually he mentioned that his left leg would not move. It was soon evident that both legs were paralyzed. He could not flex the hips or knees. There was barely perceptible dorsal and plantar flexion of the feet. Grossly, sensation to pressure and pinching appeared intact. He did not complain of parasthesias. Aware of his fluctuating chronic renal insufficiency and azotemia with rises in the serum potassium, there appeared to be little doubt that the paralysis was directly related. In the past at Togus we had encountered a patient with hypopotassemia and voluntary muscle paralysis incident to ureterosigmoidostomy[6] but up to this point we had never seen paralysis associated with hyperpotassemia despite occasional findings of ECG changes compatible with elevated serum potassium. Immediate laboratory studies disclosed the following values: venous CO_2, 34 vol. % or 15 mEq./L.; chlorides, 100 mEq./L.; sodium, 121 mEq./L.; and potassium, 8.5 mEq./L. The ECG was typical of hyperpotassemia (Fig. 1). A routine BUN earlier in the day before the advent of the paralysis was 70 mg. %. Because of the collapsed veins it was possible at first to introduce only a #22 needle but this sufficed to start an IV infusion. In the next 12 hours he received 1000 cc. of 10% dextrose in physiological saline to which was added another 50 Gm. of dextrose and 50 units of insulin, 1000 cc. of 5% dextrose in water with 10 units of insulin, 500 cc. of 1/6 molar sodium lactate solution. Beginning immediately and at 6-hour intervals he was given retention enemas of sodium polystyrene sulfonate (Kayexalate®), a cation exchange resin. This routine which was maintained for 36 hours was started with 15 Gm. of Kayexalate in 100 cc. of water initially and was increased in subsequent administrations to 30 Gm. of Kayexalate in 10% dextrose in water.

It is of interest that some minutes after the infusion was started attention was directed to the upper extremities. It was surprising to find that neither of them could be moved volun-

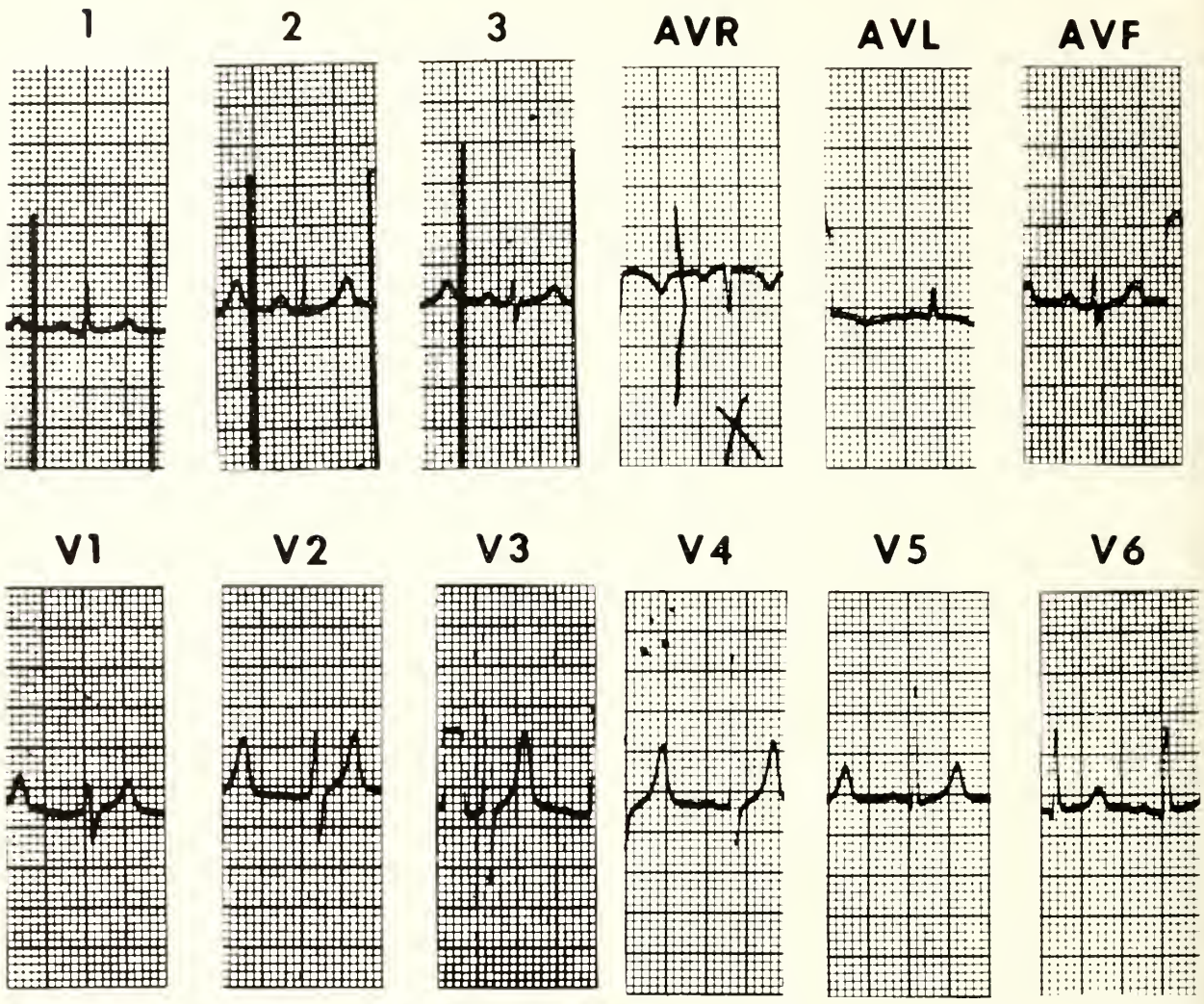


Fig. 1(b). Electrocardiogram taken Jan. 11, 1966, showing return to almost normal with residual but much less marked peaking of T waves in chest leads.

tarily and that the patient was now a quadriplegic. In the further preoccupation with introducing another needle into a vein, seeking the laboratory studies, and planning the course of treatment and expecting the asthenia to continue perhaps for at least a few hours, our gaze was not fixed upon the limbs. We mention this point because by the time we were in a position to follow him more closely at least 90 minutes had elapsed and we were again surprised to find that the upper extremities now could be moved. The same was true of the lower extremities within a half-hour. Incidentally the veins were now visible and turgid. Presumably the subtle advance of the paralysis was matched by an imperceptible reversal shortly after initiation of treatment. This seemed to liken the patient to a delicate indicator of the state of the serum potassium concentration as he swung into and out of the asthenic state.

The next morning (12 hours later) capillary blood pH was 7.27, serum potassium had fallen to 5.5 mEq./L., venous CO_2 remained at 34 vol. % or 15 mEq./L., while sodium rose to 133 mEq./L and chlorides to 106 mEq./L. The urine output was 1,375 cc. The BUN showed a rise to 140 mg. %; however, the BUN at the peak of the paralysis was not obtained and it is not known whether this represents the highest level attained. Treatment was continued with more concentrated glucose solution, insulin, and 1/6 molar sodium lactate solution. Two ampoules of sodium bicarbonate (88 mEq.) were added to the infusion. Intravenous fluids were discontinued 48 hours after the onset of the episode of paralysis when the patient had stabilized to a good oral intake of fluids. The pH now rose to a low normal of 7.36 with a further fall in the potassium level to 4.4 mEq./L. and corresponding improvement of the other electrolyte factors. Four days later the BUN fell to 60 mg. % and the serum potassium to 3.7 mEq./L.

For obvious reasons this patient with entrenched and enlarging calculi and pyelonephritis in a solitary kidney with progressive impairment of function has a dim future. He will continue to have fluctuations of the BUN and potassium levels despite good drainage and copious fluid intake. Ultimate failure seems inevitable. On Feb. 7 when he appeared weak while being examined on morning rounds, serum potassium was found to be 7.9 mEq./L. At this point he was restricted to a diet with no more than one Gm. (about 26 mEq.) of potassium daily. On Feb. 23, the potassium level was 3.9 mEq./L. and the ECG was normal. To date the potassium level, except for one occasion, has remained below 5 mEq./L. with the patient on this diet though the BUN shows wide swings above normal. An interesting finding is a persistent pH at levels just below the lower limit of 7.35 in the presence of normal sodium and chloride levels. On March 24 the pH was 7.12 and

on March 28 it was 7.16. The patient has been up and about and has no pulmonary diseases. Presumably the acidosis is due not to deficit of "fixed base" but to the retention of the phosphate, sulfate, and organic acid group of anions which is compatible with severe, renal impairment of function.

Discussion

The Symptoms: The paralysis of hyperpotassemia is described as ascending[2,4] as in our patient. There may be anxiety, apprehension, shallow respirations, listlessness, parasthesias, anesthesia pallor, vascular collapse, bradycardia, and irregular cardiac rhythm. Pain sense is said to remain intact. There may be convulsions and even respiratory paralysis.[3] Our patient had only a vague anxiety, a suggestion of an impending vascular collapse, and pallor. None of these were dramatic or obvious and would not have come to our attention were it not for his seemingly casual complaint of weakness of the left lower extremity.

Circumstances Underlying Hyperpotassemia: While potassium accumulation in the extracellular space may be present in Addison's Disease, severe dehydration, severe burns resorption of old blood, tissue breakdown, severe diabetes, and shock, and circulatory failure with their diminished oxygenation, the severe sequelae of hyperpotassemia are chiefly associated with acute or chronic renal failure with oliguria or anuria and uremic acidosis. It is difficult to produce potassium poisoning with ordinary intake of foodstuffs or potassium chloride up to 10 Gm. taken orally in one dose in the presence of normal kidneys and circulation.[2] In contrast, patients with chronic renal insufficiency which implies extreme loss of functioning nephrons, while able to secrete endogenous potassium and even exhibit urine outputs above oliguric levels, will develop toxicity with excessive exogenous intake as for example in the drinking of fruit juices or administration of medications containing potassium.[2,3,5] This was clearly demonstrated in our patient. By parenteral routes it is possible to cause intoxication in normal individuals by too rapid accumulation if administered at a rate greater than 30 to 40 mEq./hour. The daily turnover of potassium may range between 50 and 150 mEq. Eighty percent is eliminated in the urine and 20% in the stool.[2] The susceptibility to toxicity in acute or chronic failure is enhanced in the presence of hyponatremia whether due to dilution or depletion.[5]

Factors Influencing Toxicity: The cause of the paralysis or asthenia is thought to be related to altered muscle metabolism.[3] Another explanation is derived from the concepts of the manner in which striate muscle is stimulated. It is believed that the conduction of the impulse along a muscle fiber is blocked when either too much or too little potassium is present outside the cell wall.[2] In the case of hyperpotassemia it is increased. Serum levels of 10-15 mEq./L. are considered lethal but death can and does occur at lower levels. The onset of toxicity is determined by a combination of factors including the ratio of the intracellular to extracellular potassium, the degree of hyponatremia, and the extent of shift of potassium out of the cells and the degree of impairment of secretion by the renal tubules under the influence of the acidosis.[3,5] A low calcium and a high magnesium are additional factors.[2] The clinical state including the ECG changes are the result of all these ionic influences. Sodium and calcium are known to be physiological antagonists to potassium. A patient may be toxic at a level of 6 mEq./L. if the hyponatremia, hypocalcemia, and acidosis are pronounced. On the other hand a level of 8 mEq./L. may be tolerated if the sodium, calcium, and pH are normal. While the high figures have a grave implication they do not rule out reversibility of the clinical state as demonstrated by our patient. Death may follow the appearance of paralysis by minutes or hours[5] and is related to the cardiotoxicity with its disturbance of intraventricular conduction and sudden cessation of heart action in diastole.[3] The earliest typical ECG change is appearance of peaked T waves. Later as the potassium increases intraventricular block may be seen and atrial activity may cease. Loss of P waves is a very serious sign. There may be arrhythmias and ultimately a sine wave tracing may be observed.

It is paradoxical that these changes due to the accumulation of extracellular potassium may appear even in the presence of cellular deficiency of this ion.[2,8] From the aspect of diagnosis the ECG changes and the serum potassium values though not always in close correlation are complementary but the ECG is considered a highly valuable, quickly obtained index of the degree of potassium toxicity that allows initiation of early countermeasures even before all the laboratory results are obtained.

Treatment: Glucose protects against the toxicity of potassium not only by reducing the catabolism that releases the electrolyte into the extracellular space but chiefly by incorporating it with water and phosphate in the formation of glycogen. In essence this is a shift of the potassium back into the cells. Insulin accelerates this process. The combination of glucose and insulin is considered the most valuable of the effective immediate corrective measures. It is felt that in our patient this initial aspect of treatment began the surprisingly rapid reversal of the paralysis. Up to 100 cc. daily of 95% alcohol as a further caloric source to prevent protein catabolism is also advocated. Our patient was started on what amounted to a 15% dextrose solution with the insulin at a ratio of one unit to every three grams of glucose. Up to 25% dextrose solution may be given and the insulin up to one unit for every two grams of the glucose. The saline provided the water to combat the dehydration and the sodium to counter the toxic activity of the potassium and to replenish the base deficit. This was further enhanced by the use of the 1 6 molar sodium lactate solution. Ten percent calcium gluconate could have been given to some advantage as a direct antitoxic agent. Its effect however is limited and brief in duration. Two hundred cc. of 5% saline has been given as a rapid means of lowering the potassium level for a

transient effect but restraint in the introduction of sodium is advised where there is a possibility of pulmonary edema, hypertension, congestive failure, or inability to excrete sodium and water. The greatest benefit from the sodium is attained in the instances of a depleted store.

While the above measures assure the objective of quick reduction of the toxic potential of the accumulated potassium for a brief period which could last for hours, the sodium polystyrene sulfonate resin administered rectally at intervals of several hours is the means of providing a sustained, definitive removal of the potassium by the application of the principle of exchanging one ion for another. Sodium ions released into the large bowel are replaced by the accumulated potassium thereby reducing its extracellular level. This aspect of treatment is slower as compared to the other measures. Exchange ion resins may also be given orally for a few days until a potassium level of 4 to 5 mEq./L. is reached. Prolonged use of the resins however may result in excess loss of calcium and magnesium as well as potassium.[2]

While hyperpotassemia may occur in acute or chronic renal failure a distinction must be made with respect to obstructive uropathy in which once the block of flow is eliminated fluids can be given freely. Our patient fits this category. In contrast patients with acute renal failure encompassed by the term "acute tubular necrosis" or in those with hypertension or failing circulation the amounts of fluid and sodium that may be administered would be subject to sharp restriction.

Another auxiliary treatment aid that has been recommended is testosterone to prevent tissue breakdown that releases potassium. Finally the most effective means of reversing a grave level of hyperpotassemia is dialysis in one form or another, particularly in the non-obstructive types of renal failure.

Summary

We have reported another instance of transient quadriplegia of hyperpotassemia which developed in a patient with chronic calculus disease in a solitary kidney which drained only through a permanent nephrostomy. Incident to the blockage of this tube and consequent acute pyelonephritis superimposed upon a chronically infected kidney, the patient accumulated excess extracellular potassium, became hyponatremic and dehydrated. The paralysis began as a weakness in the left lower extremity and gradually ascended to include the upper extremities. The serum potassium was 8.5 mEq./L. and the ECG changes were typical of hyperpotassemia. Treatment began with the immediate administration of concentrated glucose in saline to which insulin was added. This early aspect of treatment seems to have been responsible for the rapid reversal of the paralysis. Additional measures included intravenous 1/6 molar sodium lactate solution, sodium bicarbonate, and rectal retention enemas of an exchange resin. This patient's state of renal insufficiency on the borderline of renal failure requires continued dietary restriction of potassium intake. Hyperpotassemia which is most commonly associated with renal failure whether primarily acute or secondarily in chronic renal disease like hypopotassemia can cause voluntary muscle paralysis which may be followed by death. In hyperpotassemia the immediate danger is cardiac standstill in diastole. A rapidly rising potassium serum value constitutes an emergency and calls for immediate corrective management which often can reverse progression to an otherwise fatal outcome.

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GEORGE E. SULLIVAN, M.D.

GEORGE E. SULLIVAN, M.D. of Fairfield, Maine assumed his duties as President of the Maine Medical Association at the 113th Annual Session banquet on June 13, 1966. Dr. Sullivan served as Councilor for the Fourth District from 1962 to 1965, the last year as Council Chairman and as President-Elect from 1965 to 1966.

Dr. Sullivan was born in Everett, Massachusetts on February 16, 1911, the son of Jeremiah W. and Elizabeth M. Sullivan. He was graduated from Everett High School in 1927, Boston College in 1932 and received his medical degree from Boston University School of Medicine in 1936.

Dr. Sullivan is a member of the American Medical Association, the American Society of Anesthesiologists, the Maine Medical Association and the Somerset County Medical Society.

Mrs. Sullivan is the former Ellen F. Constantine of Everett, Massachusetts. Dr. and Mrs. Sullivan have nine children: Ellen Elizabeth Currie of Anson, who has six children; William E. of North Andover, Massachusetts, employed by the Kendall Company; Lawrence W. of Sugarloaf, Kingfield, a contractor, married and has one child; George, Merchant Mariner, a graduate of Maine Maritime Academy, Castine; Richard P., employed by Abco, Poland Spring; Ann, a student at Westbrook Junior College, Portland; Barbara, a student at Coburn Classical Institute, Waterville; Jane, a student at Fairfield Junior High, Fairfield; and Mark E., a sixth grade student at Fairfield Elementary School, Fairfield.

Maine Medical Association

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Continued on Page 156



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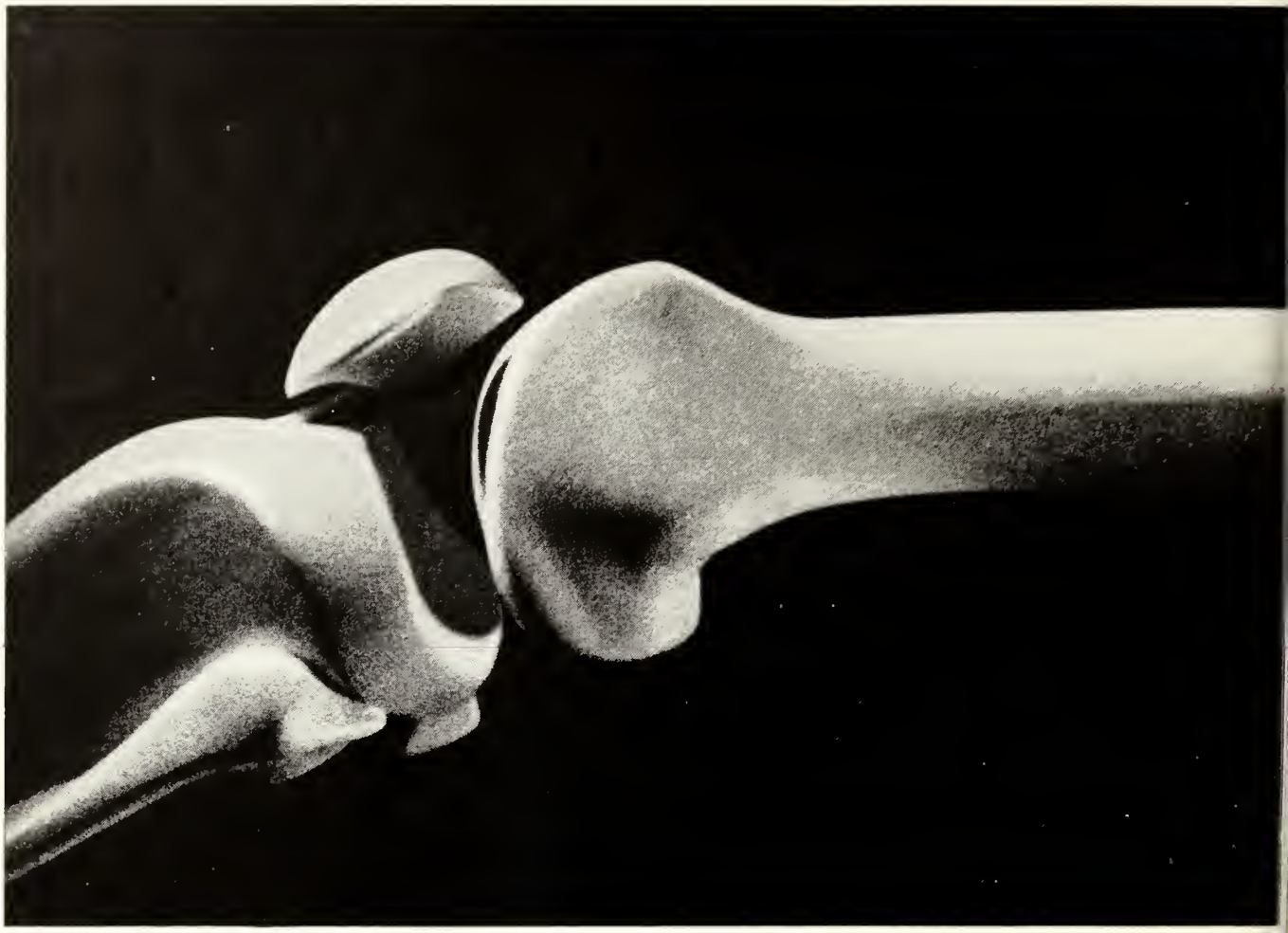
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Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should be closely supervised and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Make regular blood counts. Use greater care in the elderly.

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The FTA - ABS Test

CHARLES H. OKEY, Ph.D.*

The Fluorescent Treponemal Antibody – Absorbed test (FTA-ABS) is the latest in a series of treponemal tests developed for use in syphilis serology. It has supplanted the FTA-200 test in this laboratory because of the greater sensitivity and specificity it affords. Present indications, based on impressive evaluation statistics, are that the FTA-ABS will become the generally used treponemal test because of its ease of performance and it may replace eventually the Treponema Pallidum Immobilization test (TPI) in all but a minority of problem cases. The availability of the absorbed test now in this laboratory led us to believe it would be helpful to discuss the principle and mechanics of the test and its use in syphilis diagnosis.

Beginning with the application of the complement fixation principle by Wasserman early in this century, a number of serologic procedures were developed to assist physicians in the diagnosis of syphilis. Simpler tests, such as the Kahn, Kline, Mazzini and VDRL, used the flocculation principle to show the presence of reagin in the serum of syphilitic patients. These tests, particularly later when the more nearly defined cardiolipin antigens were used, increased the accuracy and reliability to a considerable degree. The VDRL test has become the standard STS in the United States. Reproducibility of the test from one laboratory to another over the country has reached a high degree of correlation because of the training and evaluation program of the Venereal Disease Research Laboratory, U.S.P.H.S., available to all state laboratories and through them to individual laboratories, both hospital and commercial. Despite the relative excellence of the VDRL test as a screening test applied quickly and cheaply to large numbers of specimens, it does have shortcomings, particularly the reactivity found in some serums due to what became known as the "biological false positive" (BFP). This finding may be either acute and short-lived or chronic with persistence over a long period of time. Intensive investigation of the chronic BFP, which eliminated the possibility of syphilis, indicated the possibility of association with a collagen disease. The lack of complete specificity could only be overcome by using another testing procedure employing an antigen consisting of T. pallidum. The first successful test of this type was described in 1949 as the TPI.

The TPI test is a delicate and technically exact method involving viable treponemes harvested from artificially infected animals. The test is expensive and available only in a half dozen laboratories in the country. The sensitivity and specificity are excellent and the test is considered to be the definitive laboratory method in syphilis serology. However, the expensiveness of the test stimulated the search for another less demanding method and this led to a consideration of the new field of immunofluorescence. The fluorescent antibody tests had already been applied successfully to the laboratory identification of several bacterial and viral pathogens with considerable saving of time and a high degree of accuracy. The technics are based on the conjugation of antibody molecules with fluorescein. Despite conjugation with fluorescein, antibodies retain the capacity to react with their corresponding antigens in a specific fashion. This laboratory has used the technic intensively in the direct variation of the method in which Group A beta hemolytic streptococci and rabies virus are exposed to appropriate specific conjugates following which fluorescence is observed with the microscope, using ultra-violet light illumination to excite visible secondary radiation. Other direct methods are used to demonstrate antibody interaction with known antigens.

Unidentified antibodies may be used with known antigens in what are known as indirect methods and this lends itself admirably to serologic testing such as the FTA-ABS test. With this method, serum antibody under examination is exposed to the known antigen for sufficient time to allow for union and then excess unattached antibody is removed by washing. Fluorescein-labelled antiglobulin is added and time allowed for union, followed by washing to remove excess of conjugate. In this coupling, the antiglobulin (prepared by immunizing a goat with human globulin) attaches to the human globulin traces left on the original antigen. Microscopic observation demonstrates fluorescence which proves the presence of an antigen-antibody reaction.

In the original immunofluorescence treponemal test,

*Director, Public Health Laboratory, Augusta, Maine.

nonviable T. pallidum organisms were fixed on a slide patient serum at 1:5 dilution was applied and then followed by an antihuman globulin conjugate. Patient antibodies that had combined with the treponemes reacted with the conjugate and fluorescence was observed. It was soon discovered that the rate of nonspecific reactions was excessive, reaching 20% in the patients who did not have syphilis. A dilution level of 1:200 was next selected for use and this gave good reproducibility with an acceptable balance between specificity and sensitivity. The test was widely used at this dilution level and was available in this laboratory for over two years.

During this time, parallel testing with the TPI test was carried on with aliquots of serum submitted to the Venereal Disease Research Laboratory. Correlation between the results of the respective tests was exceedingly close. However, large scale parallel testing in several laboratories having available substantial numbers of serum specimens from patients categorized by intensive clinical evaluation indicated the need for improvement in the FTA-200. A dilution of 1:200 decreased sensitivity and a dilution of 1:5 reduced specificity. Rather than using other compromise dilution levels, an absorption procedure was developed as an alternative resulting in a greatly improved modification of the basic test. Patient serum was absorbed with Reiter's treponemes, a nonpathogenic species closely related to T. pallidum, resulting in the removal of antibody normally found in humans although present in varying amounts. These antibodies result from exposure to the treponemes carried as part of the normal flora, particularly the mouth. Because these treponemes share certain common antigens, known as group antigens, with T. pallidum, significant levels of antibody to these antigens are found in many human serums and complicate the objective of specific testing. Absorption with Reiter's treponemes removes the group antibodies without significantly altering the antibodies against the specific antigens of T. pallidum. By removing the group antibodies in this manner, it is possible to dilute the serum at only a 1:5 level, thereby markedly increasing the sensitivity yet maintaining a high degree of specificity. In practice, the 1:5 dilution of patient serum is mixed with an extract of Reiter's treponemes prepared by ultrasonic disruption and placed on the fixed smear of T. pallidum. The group antibodies preferentially attach to the sonicate and the specific T. pallidum antibodies adhere to the pallidum cells. After thorough washing to remove all serum components not firmly attached to the cells, the antiglobulin conjugated with fluorescein is applied and after additional washing, the test is completed using the fluorescence microscope. Appropriate titrations and controls are carried out as part of each test run.

There are certain generalizations that may be made from experience to date with the FTA-ABS as compared to the TPI:

1. the FTA-ABS shows reactivity at an earlier stage than the TPI, being reactive in some patients during the primary stage when the VDRL is as yet nonreactive;
2. both tests remain reactive longer than the VDRL, the FTA-ABS shows greater longevity than the TPI;
3. treatment in the latent and late stages of the disease may never result in a nonreactive serologic status with either test;
4. the FTA-ABS is slightly less sensitive in detecting latent and late cases;
5. the FTA-ABS specificity may not be complete in that the collagen diseases may give reactive results in some cases.

The following suggestions are made to facilitate the mutually satisfactory use of the laboratory. The VDRL qualitative test is applied to all requests for STS with the modification that any specimen showing roughness or frank reactivity will be examined quantitatively. This approach with serums showing slight degrees of reactivity eliminates the possibility of missing a high titered serum on the basis of the prozone phenomenon. Reactive results may be followed by an additional specimen as a check test. If the results of these tests are at variance with the clinical impression, another specimen may be submitted for treponemal testing; however, a rise in titer greater than two-fold between the two tests is strongly indicative of active disease. A static titer may indicate disease or a BFP. Frequently, second tests are nonreactive, indicating the disappearance of the cause for an acute and temporary BFP. A request for a TPI test may always be made should the physician suspect latent or late disease. It should be born in mind that the FTA-ABS will remain reactive for long periods of time, particularly if the disease was treated late. The test is more indicative in treated syphilis of a history of the disease rather than active disease, as is the case of the VDRL. The VDRL is quite adequate for following the effects of therapy; the quantitative test may vary by one dilution which is within the variability of the test but a trend may be established with repeated testing. A two-tube rise in titer usually is indicative of relapse or reinfection.





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DEMETHYLCHLORTETRACYCLINE
300 mg FILM COATED TABLETS

Effective in a wide range of everyday infections—respiratory, urinary tract and others—in the young and aged—the acutely or chronically ill—when the offending organisms are tetracycline-sensitive.

Contraindication—History of hypersensitivity to demethylchlortetracycline.

Warning — In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of discomfort.

Precautions and Side Effects — Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg of demethylchlortetracycline HCl.

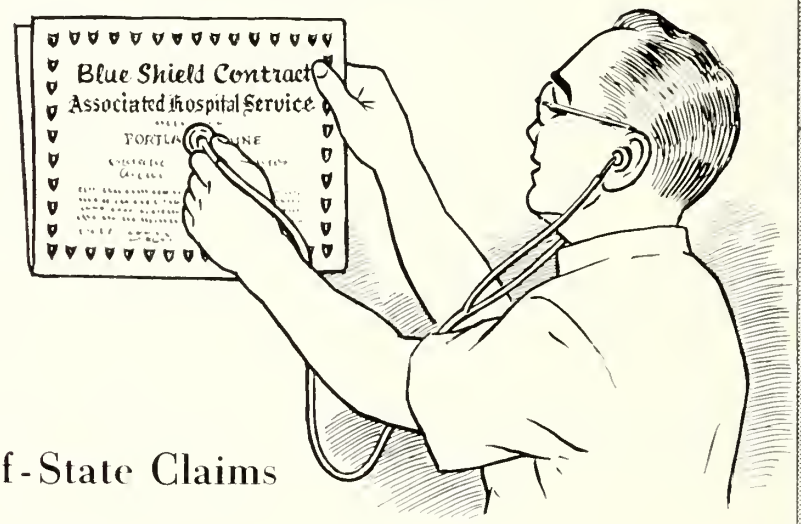
Tablets: film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

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Blue Shield Plan
OF THE
Associated Hospital Service of Maine



Out-Of-State Claims

How do I file an out-of-state Blue Shield Claim?

You can use Maine's Report to the Physicians' Service Division or request one from the Plan involved. If you use Maine's, be sure to identify the out-of-state Plan clearly on the Claim Form.

Check directories made available by A. H. S. for address of Plan and mail Claim to them.

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Maine Heart Association Notes

116 State Street, Augusta, Maine

The Search for the "Coronary" Archetype and the Damocles Test

Atherosclerosis, and especially occlusive disease of the coronary arteries, is the commonest destroyer of the American male. Epidemiology has turned technique to the discovery of the prevalence of the misadventure of acute coronary thrombosis, and myocardial infarction, arising in the course of ubiquitous vascular degeneration.

In the dismaying complexity of atheromatosis, thrombosis, and vascular vulnerability, the epidemiologist tries . . . to determine the "profile" of the victim, risk factors, the most likely elements of a formula, which, when combined, will show him the individual peculiarly susceptible to acute coronary closure. . . .

. . . no preventive regimen can be applied until these peculiarities of the "coronary prone" individual are known. It may be that even then preventive measures will be impractical.

It is signicant that many . . . coronary victims "maintained the eating habits of a robust early manhood after caloric and nutrient requirements had declined sharply."

Overweight, overnutrition, and cigarettes appear once again to be the menaces to the man with the vulnerable genetic constitution. . . .

Work . . . suggests it might be just possible that not smoking cigarettes and keeping one's weight at that of age 25 would prevent much atheromatosis, and that this is all we need to be told.

There has been shown that the almost inevitable fate of the American male is to gain weight during middle age. Reversal of this pattern would probably prolong many lives. Unfortunately, for a compelling motivation, the short life has for too long been equated with a merry one.

Submitted by Jacob B. Dana, M.D.

Reference — Editorial, Circulation, Vol. XXXIII, pages 676-678, 1966.



"All Interns are Alike"

It stands to reason. They all go through the same training; they all have to pass the same tests; they all have to measure up to the same standards; they all are underpaid, too. Therefore, all interns are alike.

That's utter nonsense, of course. But it's no more nonsensical than what some people say about aspirin. Namely: since all aspirin is at least supposed to come up to certain required standards, then all aspirin tablets must be alike.

Bayer's standards are far more demanding. In fact, there are at least *nine specific differences* involving purity, potency and speed of tablet disintegration. These Bayer® standards result in significant product benefits including gentleness to the stomach, and product stability that enables Bayer tablets to *stay* strong and gentle until they are taken.

So next time you hear someone say that all aspirin tablets are alike, you can say, with confidence, that it just isn't so.

You might also say that all interns aren't alike, either.

BAYER ASPIRIN

ORANGE FLAVORED BAYER ASPIRIN FOR CHILDREN

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Laser Beam Used in Treatment of Melanoma

A laser, which exerts enormous power in the form of coherent light, has been found to do precisely what red and infra-red rays do — but speedily and violently.

The results of this research by Drs. Kathleen Stratton, Madhu A. Pathak, and S. Fine were described recently in a report by the American Cancer Society, which helped support the work.

Several scientists have treated a pigmented cancer called melanoma with a laser beam, and they have seen the tumors sometimes disappear after laser irradiation. These spectacular demonstrations were possible only in heavily pigmented tumors; evidently something in their coloring matter responded dramatically when irradiated with a laser beam.

The pigment which imparts color not only to black cancers called melanoma but also normal skin and other colored tissues is called melanin. Melanin is a polymer made up of molecules with a tremendous capacity for joining up with similar molecules. A unique characteristic of melanin molecules is the presence of some atomic bonds with single electrons instead of the conventional pairs.

The Stratton group use a technique called E. S. R., or electron spin resonance spectroscopy which shows up unpaired electrons or free radicals, molecular fragments seeking to join up with other complete or incomplete molecules.

When a ruby laser beam irradiated dark hair or skin samples for only a few millionths of a second, it produced some vaporization of the specimens. However, the intensity of the melanin free radical signals was increased by the laser pulse in the same way as continuous red or infra-red light causes an increase. Conventional incoherent continuous light sources do not, of course, have the capacity to deliver equivalent energy in such short time periods. And as with red and infra-red continuous light, when the laser was tested against white hair and white skin, nothing happened; the signals are only produced in tissues containing melanin.

These experiments were carried out at low temperature since the radiation-induced signals, in contrast to the stable intrinsic melanin E. S. R. signal, disappear rapidly at room temperature. Ultra-violet and X radiation also induced E. S. R. signals in both pigmented and non-pigmented skin and hair at low temperature, but these were different from the enhanced melanin signal produced by red, infra-red and ruby laser light.

These basic studies are aimed at finding out how various forms of radiation affect certain biological molecules in various tissues. Such knowledge is essential for understanding and improving the treatment of pigmented tumors such as melanomas. These tumors are one of the most prevalent and deadly types of skin cancers.

American Cancer Society News Service Release

Many anxious patients need more than just calming.

Stelazine®
brand of trifluoperazine

offers true tranquilization.



Sedative or muscle relaxant-type tranquilizers are often all that's needed for patients with temporary situational anxiety. But in the many patients whose anxiety presents a continuing problem these agents are limited by their generalized dulling effects.

'Stelazine' can attack anxiety directly without producing annoying dulling effects. On 'Stelazine', patients can react more normally to day-to-day stress yet remain alert, able to carry on their normal activities.

Contraindicated in comatose or greatly depressed states due to CNS depressants and in cases of existing blood dyscrasias, bone marrow depression and pre-existing liver damage. *Principal side effects,* usually dose related, may include mild skin reaction, dry mouth, insomnia, fatigue, drowsiness, dizziness and neuromuscular (extrapyramidal) reactions. Muscular weakness, anorexia, rash, lactation and blurred vision may also be observed. Blood dyscrasias and jaundice have been extremely rare. Use with caution in patients with impaired cardiovascular systems. Before prescribing, see SK&F product Prescribing Information.

SK&F Smith Kline & French Laboratories, Philadelphia

County Society Notes

WASHINGTON

The Washington County Medical Society held two recent meetings, one on April 14, 1966 and the second on May 5, 1966. Both meetings were held at Dennys River Inn in Dennysville, Maine.

At both meetings the members discussed the impact of Medicare upon our local hospitals and its effect upon office practice. The members also discussed the new Blue Cross-Blue Shield fee schedule.

KARL V. LARSON, M.D., *Secretary*

CUMBERLAND

Fifty-five members were present at a meeting of the Cumberland County Medical Society held at Westcustogo Inn in Yarmouth, Maine on May 19, 1966.

Following a social hour and smorgasbord, the secretary's minutes were read and approved. Alvin A. Morrison, M.D. introduced the Councilor from the First District, Paul S. Hill, Jr., M.D. and one of the newer members, Vassilios Handanos, M.D. of Pineland, with his guest, Dr. Apaydin.

Dr. Morrison read a letter from Lloyd G. Davies, M.D. suggesting society opposition to three legislative bills: "The Medical Restraint of Trade Bill", the Fogarty Bill concerning adult health evaluations, and a bill concerned with unemployment compensation. Following prolonged discussion, the society voted to turn the whole matter over to Howard P. Sawyer, Jr., M.D., Chairman of the Legislative Committee of the Maine Medical Association, for evaluation and recommendations from that committee.

Dr. Asherman spoke in behalf of the Association of Medical Assistants which he has observed to be a very worthwhile organization. Albert Aranson, M.D. spoke briefly on Medicare, indicating controversy as to whether the fiscal agents are "carriers" or "intermediaries". He emphasized that the matter of customary, prevailing, and reasonable fees may be troublesome areas despite the Part B Carrier's reassuring attitude.

The society approved a request by the U.S. Public Health Service to conduct a National Health Survey in Cumberland County regarding health of adolescents.

John R. Lincoln, M.D. presented a recommendation that the society go on record as approving a resolution from the Public Relations Committee of the Maine Medical Association to the House of Delegates embodying the principle that each candidate for membership on the Public Relations Committee must be a member of the Council at the time of his election to the committee. The society approved this resolution. Charles R. Glassmire, M.D. reviewed the majority and minority reports of the Ad Hoc Committee on Osteopathy and it was voted to instruct the delegates to vote for the majority report. The society also voted to instruct the delegates to vote in favor of the recommended increase in the dues of the Maine Medical Association.

STANLEY B. SYLVESTER, M.D., *Secretary*

LINCOLN-SAGADAHOC

The Lincoln-Sagadahoc County Medical Society met at The Ledges on May 17, 1966 at Wiscasset, Maine.

John H. Reed, Governor of the State of Maine, spoke of his pleasure in spending a day among the burgeoning population of Lincoln County. He then announced the creation of an Oceanographic Committee to study the resources of the sea for the use of man. He mentioned the progress of the committee making the Feasibility Study of a Medical School in the State of Maine and spoke of the industrial growth throughout the state.

The proposed 1967 budget of the Maine Medical Association was outlined and the probability of an increase in state dues was discussed. It was voted that this society instruct its delegates to approve the proposed budget and increase in dues.

A digest of the report of the Ad Hoc Committee on Osteopathy was given and a motion that the delegates be instructed to approve the report of the committee was carried.

Dr. Boswick reported the death of Arthur A. Nichols, M.D. on May 14, and the fact that he had written a letter of condolence to Mrs. Nichols.

Edmund N. Ervin, M.D. of Waterville spoke on the work of the Advisory Committee on the Care of the Mentally Retarded in this State.

GEORGE W. BOSTWICK, M.D., *Secretary*

KENNEBEC

A meeting of the Kennebec County Medical Association was held at the Augusta Country Club in Augusta, Maine on May 19, 1966. The meeting was called to order by the President, Allan J. Stinchfield, M.D.

Perry J. Culver, M.D., of Boston, Massachusetts gave a short talk on Medical Education. He discussed the trends in medical education and stressed the idea of teaching medical students concepts of learning so that they may adapt as medicine progresses.

LUCIEN F. VEILLEUX, M.D., *Secretary*

News and Notes

Department of Health and Welfare Division of Maternal and Child Health Including Services for Crippled Children (By Appointment Only)

Orthopedic Clinics

Augusta — Augusta General Hospital
1:00 p.m.: Aug. 25
Bangor — Eastern Maine General Hospital
9:00 a.m. and 1:00 p.m.: July 21, Sept. 22
Half-day Sessions 1:00 p.m.: Aug. 25
Fort Fairfield — Community General Hospital
9:00 a.m. and 12:30 p.m.: July 11, Sept. 12
Fort Kent — Peoples Benevolent Hospital
10:00 a.m.: Sept. 13
Houlton — Aroostook General Hospital
9:00 a.m.: July 13
Lewiston — Central Maine General Hospital
9:00 a.m.: July 15, Aug. 19, Sept. 16
Portland — Maine Medical Center
9:00 a.m.: July 11, Aug. 8, Sept. 12
Presque Isle — Arthur R. Gould Memorial Hospital
9:00 a.m. and 12:30 p.m.: July 12, Sept. 14
Rockland — Knox County Hospital
1:30 p.m.: Aug. 18
Rumford — Community Hospital
1:30 p.m.: Sept. 21

Cleft Palate Evaluation Clinics

Portland — Maine Medical Center
10:00 a.m.: Sept. 13

Clinics for Mentally Retarded Preschool Children

Waterville — Thayer Hospital
9:00 a.m.: July 20, Aug. 3-17-31, Sept. 7-21
Lewiston — Central Maine General Hospital
9:00 a.m.: July 11, Aug. 8, Sept. 12
Lewiston — St. Mary's General Hospital
9:00 a.m.: July 25, Aug. 22, Sept. 26

Cardiac Clinics

Bangor — Eastern Maine General Hospital
9:00 a.m.: July 22, Aug. 12-26, Sept. 9-23
Portland — Maine Medical Center
9:00 a.m.: Every Friday (holidays excepted)

Pediatric Clinics

Bangor — Eastern Maine General Hospital
1:30 p.m.: July 22, Aug. 26, Sept. 23
Fort Kent — Peoples Benevolent Hospital
10:00 a.m.: July 27
Presque Isle — Arthur R. Gould Memorial Hospital
1:30 p.m.: Sept. 28

Cystic Fibrosis Clinics (In conjunction with the CMGH and MMC)

Lewiston — Central Maine General Hospital
9:00 a.m.: Aug. 5, Sept. 2
Portland — Maine Medical Center
9:00 a.m.: July 19, Aug. 16, Sept. 20

Book Reviews

Respiratory Care, by H. H. Bendixen, L. D. Egbert, J. Hedley-Whyte, M. B. Laver and H. Pontoppidan. From the Respiratory Unit and the Anesthesia Laboratory of the Harvard Medical School at the Massachusetts General Hospital, Boston, Massachusetts. Published by the C. V. Mosby Company, St. Louis, 1965. Pages 252, Price $15.00.

"Respiratory Care" is an unusually lively book that applies the principles of respiratory physiology to the total care of the patient with respiratory problems. Lung physiology is well introduced with many excellent illustrations and diagrams that can be understood by the physician who is not primarily a respiratory physiologist, but also has many graphs and descriptions that stimulate the expert. The unavoidable physiological abbreviations are always well defined and explained. Thus the book discusses oxygen transport, physiological dead space, physiological shunting, hypoxia, hypercapnia, hypotension, dyspnea, patterns of ventilation, relation of ventilation to perfusion, ventilatory reserve with ease. It then turns to diagnostic techniques with excellent chapters on blood-gas measurements, acid base balance, and clinical diagnosis. These chapters contain again many valuable graphs, photographs of equipment and price-

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AN ANCHOR TO WINDWARD

less normograms that can easily be reduplicated for hospital use, as they are on single pages.

The next part deals with prevention of respiratory complications, including chapters on chest physical therapy and humidification. There are pertinent photographs illustrating chest physical therapy. Then follows a detailed account of therapeutic measures, including endotracheal intubation, tracheostomy, emergency resuscitation, oxygen therapy, and a most important, thorough account of the management of patients undergoing prolonged artificial ventilation. Finally there follow special problems of respiratory failure such as chest injury, intracranial lesions, myasthenia gravis, acute idiopathic polyneuritis, tetanus, poliomyelitis. There are excellent accounts of the respiratory management of acute poisoning and acute exacerbation of obstructive pulmonary disease. There are also chapters dealing with thoracic and cardiac surgery, and respiratory problems in obesity. The individual problems are discussed in detail and contain many useful therapeutic recommendations, some of which are illustrated with photographs. Appropriate case reports are included. Much care is expended on a thorough bibliography which alphabetically lists 463 references. Some chapters such as those on humidification and aerosol therapy, and emergency resuscitation will no doubt be expanded in future editions.

This is a thoroughly enjoyable text. It conveys the multiple authors' enthusiasm, resourcefulness, and genuine compassion to truly difficult problems. It is an unusual book, which is lucid to the end, in a field not noted for its clarity. Most important it conveys the physiological discipline needed to approach the management of respiratory failure. This text should become required reading for anesthesiologists, chest physicians, and cardiologists. The book is well bound, very well illustrated, has a handsome clear type, and is highly recommended.

PETER F. LANSING, M.D., Augusta, Maine

Heritable Disorders of Connective Tissue, by Victor McKusick. Third Edition, Published by the C. V. Mosby Company, St. Louis, 1966. Pages 499, illustrated, Price $18.50.

The third edition of Dr. McKusick's book is a worthwhile addition to our library. He has accomplished a difficult task by organizing this complicated subject and presenting it in easily understandable and well-readable material accompanied by many photographs and tables. The chapter on mucopolysaccharidosis and the compiled data on other possibly genetic disorders involving the connective tissue are remarkable features of this treatise.

The detailed world bibliography at the end of each chapter imparts additional value, by rendering this work an easily available source of reference to those who are interested in this subject.

It is felt that this work is valuable to many divisions of medical science and practice.

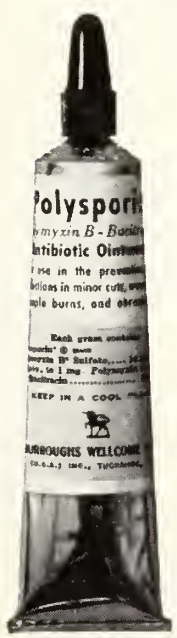
ERWIN G. RERRICK, M.D., Pownal, Maine

Hashish: Its Chemistry and Pharmacology, Ciba Foundation Study No. 21. Published by Little, Brown and Company, Boston, 1965.

Ciba Foundation has entitled its latest volume, Study Group 21, "Hashish: Its Chemistry and Pharmacology." Hashish – oriental bazaars, plaintive oboe sounds – is richer than its synonym "Marijuana," degraded by chin-tufted guitar strummers of subterranean San Francisco.

Over the years, one has had a faintly morbid interest in drug addiction and Hashish, but never to the extent of basic chemistry. This lukewarm approach has been shared by the world's researchers, and the Index Medicus to date shows but four

in treating topical infections, no need to sensitize the patient



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Caution: As with other antibiotic products, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs. **Contraindication:** This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.
Supplied: In ½ oz. and 1 oz. tubes
Complete literature available on request from Professional Services Dept. PML.

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N.Y.

papers. Cannabis, the next alphabetically listed subject, equally obscure, weighs in with thirty articles.

So much for background: for those wishing to be up to the minute on Hashish we recommend this short book, wherein are recorded the ideas of leaders in the field, who met for a day's round table discussion. It is full of titillating facts: for example, did you know that Hashish is a wide-spectrum antibiotic? The only marketing problem is how to take the pleasure out of it. Then there is the difference between the Hashish group and the Morphine circle. The Hashish people are gregarious, getting together for a smoke. They have erotic fantasies but are rendered too weak to do anything about them, too rapturous to be frustrated. The Morphine men walk alone. They like to take their dope in isolation. Paradoxically, they report no enjoyment from their opium, only a craving. Hashish apparently unleashes one's criminal tendencies. Herein lies its danger, for, oddly enough, Ciba Foundation reports it as not being a drug of addiction.

ANTHONY BETTS, M.D., Portland, Maine

STANDING COMMITTEES — 1966-67

Continued from Page 140

Carlton E. Swett, M.D., Fairview Hospital, Skowhegan (2 yrs.) – (Somerset)
George L. Temple, M.D., Fahey St., Belfast (1 yr.) – (Waldo)
John Kazutow, M.D., Box 113, Columbia Falls (2 yrs.) – (Washington)
Roger J. P. Robert, M.D., 331 Main St., Saco (1 yr.) – (York)

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Maine Society of Anesthesiology – Clement S. Dwyer, M.D., 205 French St., Bangor
Maine Chapter, American Academy of General Practice – John D. Denison, M.D., 105 Brunswick Ave., Gardiner
Maine Society of Obstetrics and Gynecology – E. Allan McLean, M.D., 29 Deering St., Portland
Maine Chapter, American Academy of Pediatrics – Everett A. Orbeton, M.D., 131 Chadwick St., Portland
Maine Society of Internal Medicine (Includes Medical Specialty Group) – Albert Aranson, M.D., 39 Deering St., Portland
Section on Ophthalmology of the M.M.A. – Jay K. Osler, M.D., 74 Birch St., Bangor
Maine Radiological Society – John F. Gibbons, M.D., 22 Bramhall St., Portland
Maine Chapter, American College of Surgeons – John F. Reynolds, M.D., 216 Main St., Waterville
Maine Trauma Committee – Frank B. Bull, M.D., Hasson St., Hallowell
Ear, Nose and Throat Group – John E. Whitworth, M.D., 336 Mt. Hope Ave., Bangor
Maine Society of Pathologists – Franklin F. Ferguson, M.D., 22 Bramhall St., Portland
Neurology and Neurosurgery Group – Daniel A. Rock, M.D., 477 Main St., Lewiston

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Delegate and Alternate Delegate to A.M.A.

(Jan. 1, 1965 to Jan. 1, 1967 & Jan. 1, 1967 to Jan. 1, 1969)

Delegate – Paul H. Pfeiffer, M.D., 14 Gilman St., Waterville
Alternate Delegate – George W. Wood, III, M.D., 156 No. Main St., Brewer

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Contraindications: In hyperexcitability and in agitated prepsychotic states. Previous allergic or idiosyncratic reactions.

Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised; especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of pre-existing symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dose—operation of motor vehicles, machinery or other activity requiring alertness should be avoided. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia, the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor, and respiratory collapse.



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By providing combined anorexigenic-tranquilizing action, BAMADEX SEQUELS Capsules help your nonshrinking patients to establish new patterns of eating less. The amphetamine component suppresses the appetite, while the meprobamate helps allay nervousness and tension. And for most patients, the *sustained* release of the active ingredients makes possible convenient one-capsule-a-day dosage.

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In the management of your hypertensive patients, Naturetin is good therapy to start with, good therapy to stay with.

In mild hypertension, Naturetin lowers blood pressure gradually toward normotensive levels. In long-term therapy, Naturetin may keep blood pressure low—for months, sometimes years. When used in combination with other antihypertensive agents, blood pressure often falls further—and lower doses of both drugs are usually possible.

Clinical trials have proven Naturetin effective—without serious side effects.[1,2] And, when used to treat patients with cardiac edema and hypertension, "in no instance did the concentration of serum potassium fall below 3.1 mEq. per liter."[3] (Normal range for serum potassium: 3.5-5.0 mEq./liter).[4]

When readings indicate hypertension, start with Naturetin, stay with Naturetin.

Contraindications: Severe renal impairment; previous hypersensitivity.

Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available—Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

References: 1. Telfeyan, S. A.: Clin. Med. *70*:1668, 1963. 2. Shepard, H. L.: J. Am. Geriatrics Soc. *11*:363, 1963. 3. Cummings, D. E.; Goodman, R. M., and Steigmann, F.: J. Am. Geriatrics Soc. *12*:161, 1964. 4. Castleman, B., ed.: New England J. Med. *268*:1462, 1963.

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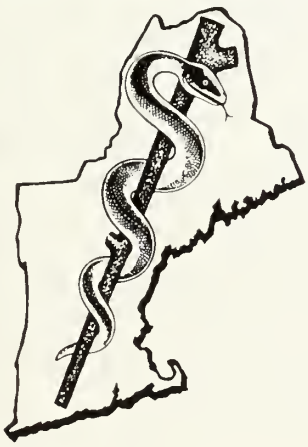
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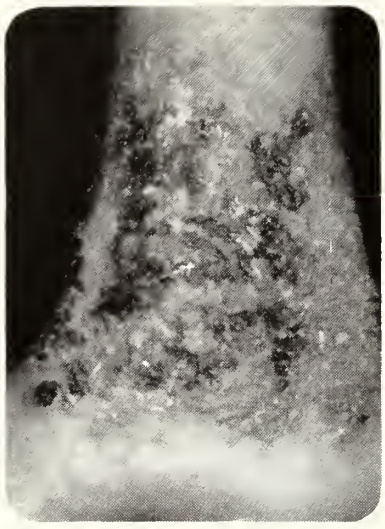
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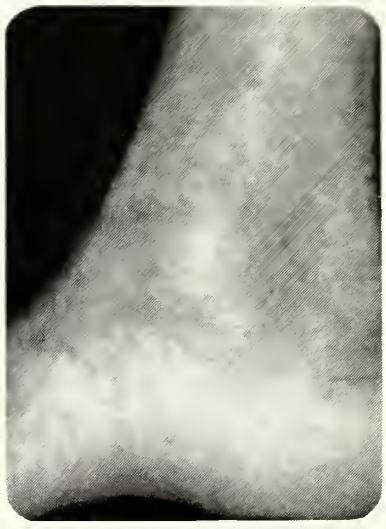
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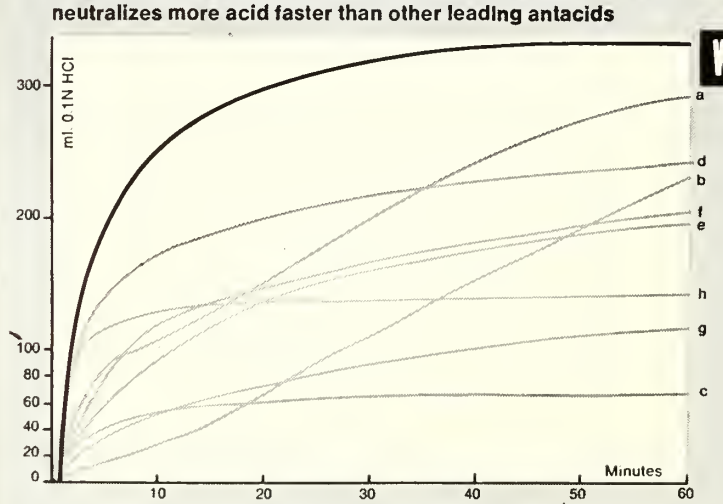
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this issue: emergency anesthesia and the common cold

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Barry Belonsky, M.D., F.A.C.A.
Staff Anesthesiologist, Hospital of The Albert Einstein College of Medicine, New York City



Medical facilities are often presented with unfamiliar patients who have unknown health histories. This is particularly true in emergency situations that arise due to accidents or acute illnesses. These cases may need prompt care requiring anesthesia, and if they involve colds, nasal allergies or other upper respiratory infections, can account for many complications which make up a major hazard during emergency anesthesia.

Administration of general anesthesia to a patient with a cold or upper respiratory infection is a hazardous undertaking. It should be avoided if at all possible. Indeed, the presence of U.R.I. is good reason for postponement of elective surgery.[1] In emergency surgery, regional or local block should be considered, but if general anesthesia is mandatory, it should be approached with utmost caution.

Since the attitude of "emergency surgery — hurry" has been replaced by "emergency surgery — watch out",[2] a knowledge of the complications is a great help in preventing them. Here is a brief outline of the problems involved and their treatment. Prevention of the complications is discussed later.

Complications during the induction of anesthesia

Most of the complications are a direct result of secretions and some a result of accompanying secondary infection. For example, *airway obstruction* due to excessive secretions occurs very commonly and is the direct effect of the cold. Respiratory exchange may be obstructed at any time during anesthesia because of excessive secretions, but is most likely to occur during induction. Suction apparatus must be available to overcome this.[3]

Excess secretions which stimulate and irritate the epiglottis and vocal chords can cause *laryngeal stridor and obstruction*. This can lead to complete laryngeal closure with resultant anoxia and death.

Bronchospasm and laryngospasm can result from secretions penetrating the bronchi and bronchioles. In laryngospasm, there are both inspiratory and expiratory stridor and difficulty in inflating the chest; in bronchospasm there is an expiratory wheeze, but not as much difficulty in inflation, although some resistance may be felt. Stridor is due to partial or complete closure of the vocal cords in spasm and the "crowing" sound is almost pathognomonic.

Secretions obstruct the nasal airways. This produces *difficulty in ventilation* through the mouth until the patient is deep enough to place an oral airway. An intravenous agent can be given to facilitate the induction of anesthesia.

Difficulties can arise if intubation is performed to ventilate the patient. For example, teeth can be broken by too vigorous attempts at intubation, or the intubation itself may be technically difficult due to secretions obstructing the view of the glottis. The postoperative sequelae of intubation ranges from mild laryngitis to pneumonia with atelectasis, and are seen far more commonly in patients suffering from colds than in normal patients.



Successive stages of laryngospasm which produce the characteristic stridor or "crowing" sound.



Progression of bronchioles into bronchospasm.

Complications during the maintenance of anesthesia *Bronchospasm* can occur in an unintubated patient due to secretions entering the bronchial tree from above, and acting as an irritant to the bronchi and bronchioles. Secretions accumulate quickly and the patient has to be suctioned continually. The whole cycle of coughing, bucking, laryngospasm and bronchospasm may ensue. The difficult decision here is whether it is better to suction the patient continually or to use an endotracheal tube which protects the cords and bronchi but introduces the risk of attendant complications.

Postoperative complications Postoperatively, complications can be more serious than even the intra-anesthesia complications, and occur much more frequently in a patient who has been intubated.[4]

Sore throat and pharyngitis can result both from the preoperative upper respiratory infection and from the drying of the mucous membranes which occurs during anesthesia.

Tracheitis and bronchitis often result from secretions trickling down the tracheobronchial tree.

Laryngitis is frequently seen in patients with upper respiratory infections who have been intubated. There is a significant increase in the incidence of laryngitis compared to that in patients without upper respiratory infections.

Subglottic edema is a condition which occurs mainly in children who have been intubated. This pathology results from an exudate developing in the areolar tissue just below the cords. Because of the small size of the child's trachea, even a 1 mm increase in size of the mucous membrane can severely impair the air passage. Children exhibit this by severe expiratory stridor and may even become cyanotic. This may so severely embarrass the child's breathing that it must be treated vigorously. Most authorities agree on the treatment[5,6,7,8] consisting of a high oxygen concentration in the inspired air (60%), plus high humidity (close to 100%). Adequate parenteral fluid intake and slight cooling of the body temperature (by a cooled oxygen tent) also help in mild cases. In severe cases, there may be hypoxia which increases the restlessness and the oxygen demand rises. Sedation is often necessary, although concomitant depression of the respiratory center is undesirable. An antihistaminic accomplishes this purpose well, and adds sedation. Since there is always a possibility that an allergic response plays a role in edema, some relief of the respiratory distress may occur. Steroids should be used to control inflammatory and allergic phenomena and swelling. If all this fails, and the patient is still restless and hypoxic, a tracheostomy should be performed immediately.

(concluded on following page)



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Cross section of trachea showing subglottic edema and lumen reduction due to mucous membrane congestion.

Pneumonia may also follow anesthesia administered to a patient with a cold. This can be caused by accumulated secretions becoming secondarily infected and causing consolidation of the lung. *Atelectasis* of the lung can result if one of the bronchioles becomes plugged by secretions, preventing aeration of the distal part of that lung. This is seen more frequently following upper respiratory infection because dry anesthetic gases aggravate the infection, causing secretions to change from watery to thick and viscid, and consequently difficult to suction.

Prevention of complications The first rule to prevent complications, of course, is to use a regional or local anesthesia whenever possible. But when emergency surgery is a must, in spite of the presence of a cold, allergy, or upper respiratory infection, here are some ways to prevent complications.

Give nose drops preoperatively. This can help shrink the congested nasal mucous membranes and reduce secretions for better air passage. (Results of this method are sometimes unsatisfactory because of the short duration of effect or rebound congestion.) For longer effect, oral antihistamines with nasal decongestants are often given to provide and maintain a drying effect on secretions.

To clear the tracheobronchial tree, instruct the patient to cough preoperatively. Cold steam or water nebulizers effectively humidify the nasal, pharyngeal and bronchial passages and often make the patient more comfortable. Tenacious secretions become more watery under humidification, clear more thoroughly preoperatively and are more easily suctioned from the airway during anesthesia.

Give intravenous fluids to those patients who appear dehydrated due to a cold. In a well hydrated patient the respiratory tract secretions are less viscid and more watery. This is particularly true in asthmatics.

Summary: Administration of emergency anesthesia to a patient with a cold or upper respiratory infection can lead to a chain of events that may result in increased postoperative morbidity and even death. This is because of the excess secretions formed in these conditions. Preoperative measures to prevent or reduce these secretions should be undertaken and will result in smoother and safer anesthesia.

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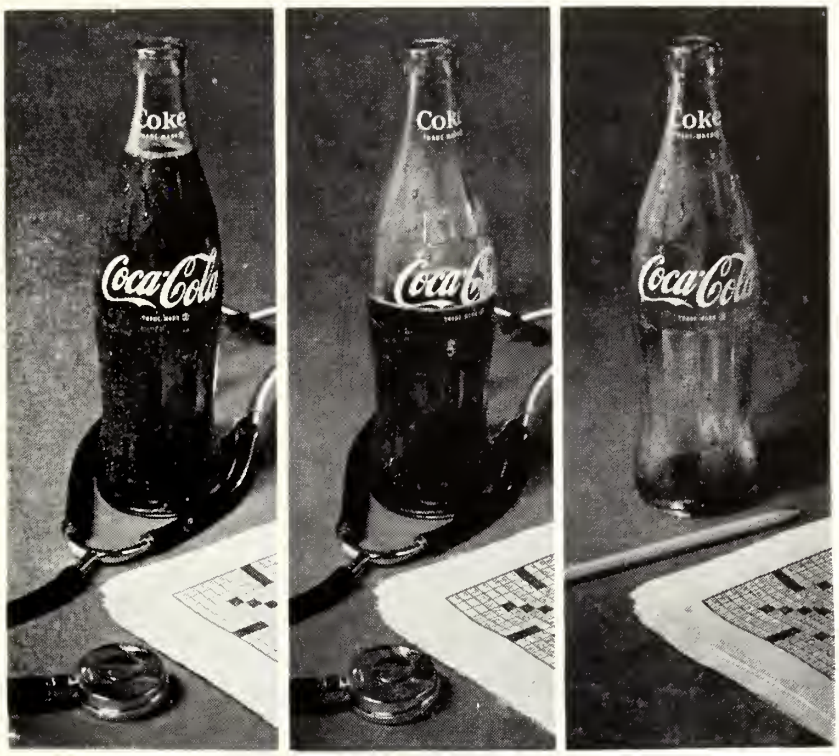
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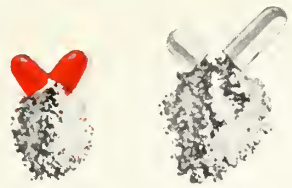
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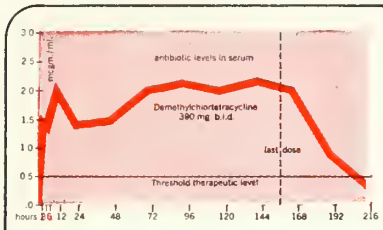




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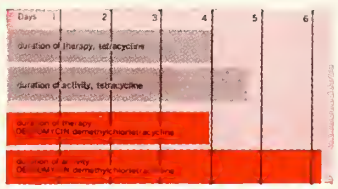
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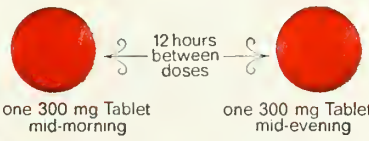
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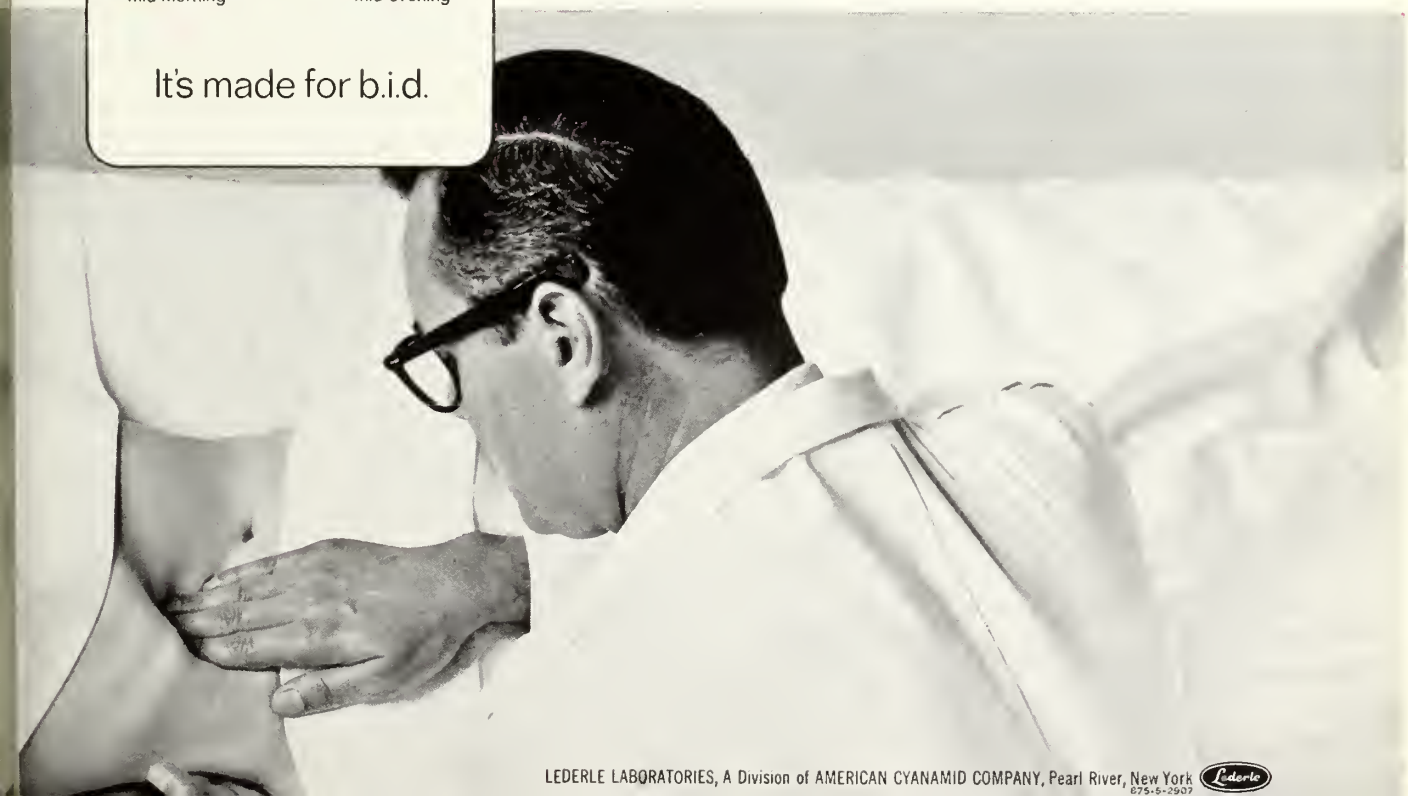
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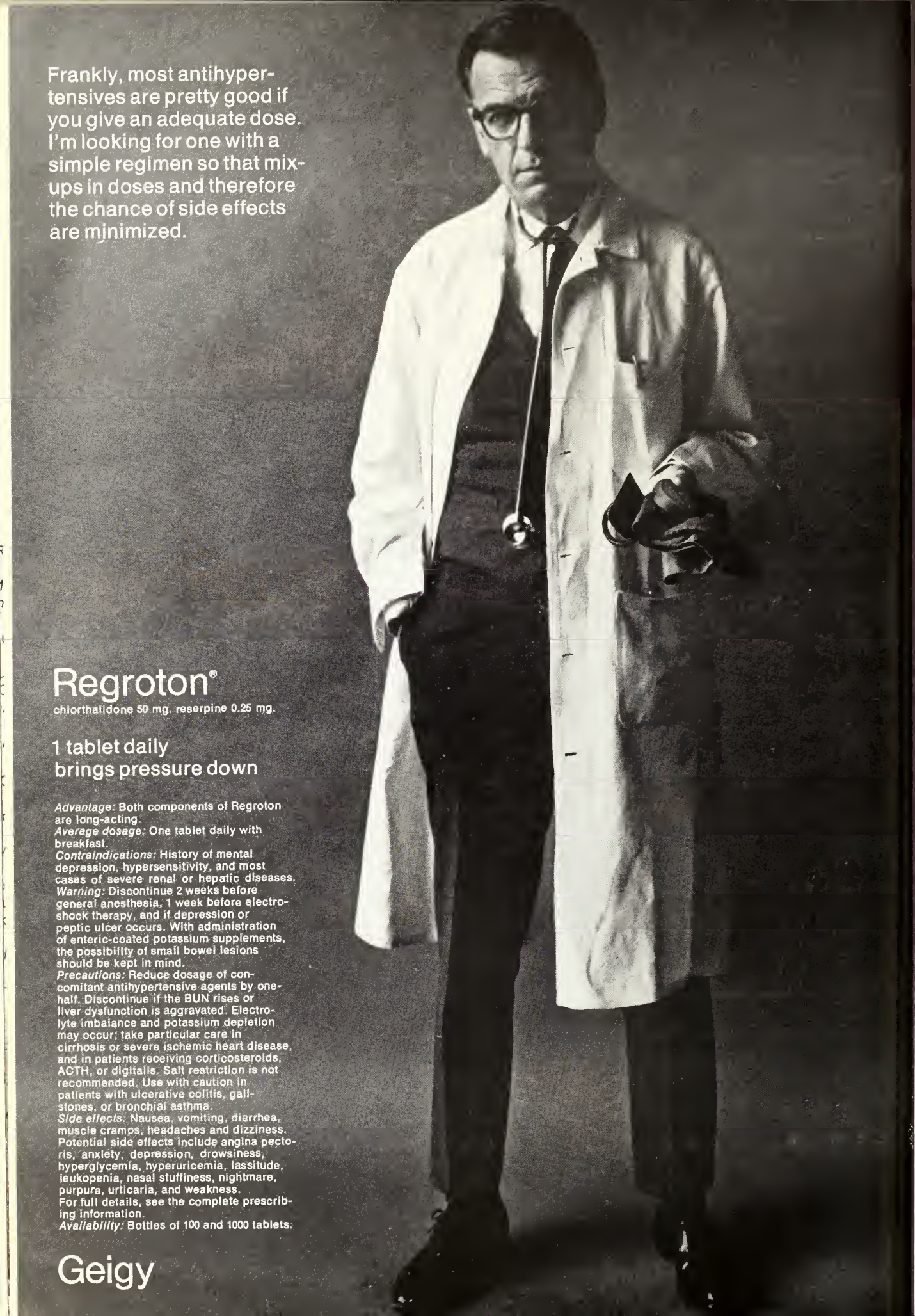
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(see previous page)

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Chondrosarcoma

John H. Shaw, M.D. and Jose Castellanos, M.D.

Chondrosarcoma, recognized during the past 30 years as a separate chondral origin, is the largest and most important group of malignant cartilage tumors of bone. Unusual sites include lumbar spine, sacrum, hand, and foot. Average age incidence is between age 30 and 60, and as opposed to the osteosarcoma, they are very slowly growing and metastasizing.

Tumors of this category are grouped in two general classifications. The first, or primary chondrosarcoma, is according to the classification of Stein, Stein, and Beller. These usually occur in young adults and children and are quite rare. Secondary chondrosarcoma are usually felt to arise from a preexisting endo-enchondroma or an osteochondroma. This tumor, more common in frequency, usually occurs in middle or adult life. Ackerman and Spjut have found in their series; however, that they have been unable to trace malignant chondrosarcomas to a preexisting nonmalignant lesion.

The clinical aspects of the tumor are divided into two groups; those which occur centrally within the skeletal system, or long bones, and those which occur adjacent to the surface of, or the skeletal structures, or soft tissue of the joints. Amputation is mandatory in the central occurring chondrosarcoma for adequate treatment. Their occurrence is usually local and x-ray appearance of the structure involved should be no deterrent to definitive surgery as the lesion is most almost well circumscribed and excision is fascial.

The growth specimens usually resemble recognizable cartilage with a white or bluish mass. They are malignant, but usually show definite encapsulation and are conscpicuous by their lobular pattern. There is usually diffuse discoloration from degeneration and hemorrhage in the larger of the tumors and myxomatous degeneration resembling a slimy mass with irregular calcification is often the rule. Microscopically, according to Lichtenstein, chondrosarcoma shows grossly proliferating cartilage but never shows neoplastic osteoids. This point is used in the most important differential diagnosis and it consists of clear separation between osteogenic and chondrogenic sarcomas. The metastases which do occur are usually by route of the central venous channels and have been noted on several cases to be metastases in continuous chains from the site of origin to the heart and pulmonary vessels. Treatment consists of radical surgical extirpation or amputation of the extremity involved and removal of the bone area of origin in the case of peripheral occurring lesions.

Case Report

A 57-year-old white male was first seen and evaluated medically at the Augusta State Hospital in August of 1948, at which time the diagnosis of Dementia Praecox, Paranoid Type was made. Admission physical examination at that time was within normal limits except for the presence of an old right lower quadrant incision. Routine laboratory work, including blood count, serology and urinalysis, were negative, and physical examination revealed no presence of masses at that time.

The patient was discharged in May of 1949 and was seen and evaluated at a later date at a clinic in Massachusetts. The patient was admitted to the New England Deaconess Hospital in September, 1952. At that time the paient underwent surgery for removal of a mass overlying the left lower quadrant of the sacrum, which was found to be attached to the gluteus maximus muscle. Pathological report on this specimen on removal was chondrosarcoma, low grade. Follow-up x-rays at that time revealed the presence of a small portion of tumor remaining at the pedicle site at the lower quarter of the lateral side of the sacrum. The patient was readmitted on November 4th for a secondary removal of the pedicle and a portion of the sacrum and coccyx. Postoperative x-rays at that time revealed a resection of the small segment of the 4th sacral vertebra to the level of the foramen.

The patient was readmitted to the Augusta State Hospital on March 13, 1960 with evidence of an old healed surgical scar on the left buttocks, approximately 16 inches in length. At this time a mass was present, measuring 35 by 50 by 12 cm. Surgery was refused at this time. The patient was kept on custodial and outpatient visit care from March 30, 1960 until January 5, 1965 when the patient developed fever, ulceration, and drainage from the mass on the left buttock, which now measured 40 cm. in length, 34 cm. in greatest diameter. At this time the patient developed diabetes and was found to have a fasting blood sugar of 260. Culture of the ulceration of



Fig. 1. Chondrosarcoma 1 month prior to surgery.



Fig. 2. Buttocks 14 months postoperative removal chondrosarcoma.

the tumor revealed staphylococcus aureus coagulase positive and Aerobacter aerogenes. The patient was placed on insulin and Orinase therapy for control of his diabetes; antibiotics for control of his ulceration and abscess. On January 25, 1965 a radical excision of the mass of the left buttock was performed. The patient was placed in a prone position. The anterior portion of the tumor rested against the greater trochanter posteriorly. The gluteal muscles were found to be markedly separated, spread and thinned out by the tumor mass pushing them medially, anteriorly, laterally. The tumor mass was separated from the attenuated normal structures without difficulty and was found to be densely adherent only to the lateral portion of the left posterior sacrum. In this area an exostosis appeared to be the route of the tumor and was rongeured away to remove all traces of parent pedicle. The main vascular supply of the tumor came through the sciatic notch above and lateral to the sciatic nerve.

Postoperatively the patient was placed on negative pressure wound suction, no grafting was necessary. The patient received 1,000 cc. of whole blood in the operating room during the procedure. A fever of 103° orally was noted on 14th postoperative day and incision of a large wound abscess was performed. The abscess was cultured and the patient was started on appropriate chemotherapy and made an uneventful recovery with primary healing of the drained area. The pathological specimen weighed 27 pounds at the specimen table and microscopically was diagnosed as a low grade chondrosarcoma. During the 14 months since operation the patient has been active, well, walking from 3 to 5 miles daily without ill effects. Present examination reveals no evidence of recurrence, the patient has no detectable limp, and there is no x-ray evidence of recurrence of calcification or exostosis.

Summary

The case of a 57-year-old mental patient with prolonged growth of a low grade chondrosarcoma with secondary excision following 2 previous operations over a 14-year period is presented. A plea is made and attention drawn to the the fact that because of the relatively benign primary diagnosis, patients can be afforded extremely significant symptomatic relief from removal of large recurrent masses without resort to crippling radical operations or severe operative morbidity with adequate separation and negative studies of areas subjacent to the tumor mass. Amputation or extirpation of bones involved with central occurring chondrosarcomas still is necessary.

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Dr. Castellanos, Augusta State Hospital, Augusta, Maine



Pelvic Arteriovenous Fistula

Report of a Case and Management

ROBERT L. CALLAHAN, M.D.*

Arteriovenous fistulas are divided usually into two types: traumatic (acquired) and congenital. Traumatic fistulas follow injury, usually missiles or knives causing wounds, and are usually found in extremities, though visceral thoracic and cervical are occasionally encountered. Congenital fistula result from failure of differentiation of the common embryologic anlage into artery and vein. Most congenital arteriovenous fistulas occur in extremities, although intracranial lesions have been reported.

The following case is presented but it cannot be said with certainty whether it represents the congenital or acquired type.

CASE REPORT

A 39-year-old white gravida II, para II female was seen in consultation in the office three weeks prior to admission to the Augusta General Hospital. She was referred by her obstetrician. For the five to six weeks prior, she had noted progressive daily constant pain in the left lower quadrant. The pain was unrelated to gastrointestinal function or urinary tract function. For three weeks she had been kept awake at night by the feeling of "her heart pounding" in the left lower quadrant. She noted progressive generalized fatigue and irritability.

PAST HISTORY AND SYSTEM REVIEW

An asymptomatic small interatrial septal defect had been diagnosed seven years ago by a cardiologist.

From age 29 through 31 several D & C's were performed by various physicians for profuse menstrual bleeding. At age 31, during a D & C, her uterus was described as normal size, and no mention of any pelvic abnormalities were made. The diagnosis of hyperplastic endometrium was made.

During early 1960, at another hospital, a caesarian section was performed for placenta previa. A normal child was delivered. On December 12, 1961 at the Augusta General Hospital a low transverse caesarian section was performed through the scar of the previous section. At this time the obstetrician described "markedly dilated and varicosed veins measuring approximately three to four centimeters in the region of the broad ligament and round ligament." A viable infant was delivered. Postoperatively the patient developed afibrinogenemia which responded well to intravenous fibrinogen. Placenta previa not present.

On December 26, 1963, because of profuse vaginal bleeding for twelve days, refractory to intravenous Premarin®, a D & C was performed. The adnexa were described as normal at this time. The uterus was enlarged. Proliferative endometrium was the pathological diagnosis and adenomyosis established clinically.

During January 1964, she was readmitted with profuse vaginal bleeding. Complete physical examination at this time disclosed a grade II systolic murmur in the third left interspace, noted on prior admissions, an enlarged uterus, and unremarkable adnexa. No thrills or bruits elicited.

On January 6, 1964 a total hysterectomy was attempted for what ultimately proved to be marked adenomyosis of the uterus. During the hysterectomy on the posterior surface of the lower uterine segment at the peritoneal reflection were "tremendously dilated engorged plexus of veins, measuring the size of a man's finger." These dilated vessels extended into the broad ligament. Severe hemorrhage was encountered and a supracervical hysterectomy was performed. Postoperatively an I.V.P. showed some holdup on the left but complete emptying, and a cystoscopy showed a large extrensic mass pressing on the left side of the bladder. She was soon discharged, asymptomatic.

On January 4, 1965 she was admitted to the Augusta General Hospital with the afore mentioned complaints. Positive findings on physical exam: a grade II pulmonary ejection murmur, healed scars in the abdomen, blood pressure 128/74, pulse 80, auscultation over the left lower quadrant of the abdomen revealed a machinery murmur, unaffected by compression of the femoral vessels at the groin, and partially damped by compression of the abdominal aorta through a moderately large panniculus. Pelvic exam disclosed a large soft tissue, compressible mass occupying the left lateral vaginal wall and vault. By placing a Bell stethoscope directly in the vagina, a loud grade V machinery murmur was heard. The extremities were negative. There were no signs of congestive heart failure. The admitting diagnosis was arteriovenous fistula, left hypogastric artery-cervical vein, etiology uncertain. Laboratory work was unremarkable. I.V.P. normal. Percutaneous lumbar aortogram disclosed the splenic artery, rapid aortic run off and opacification of a large confluent 8 x 5 cm. vascular mass density in the left pelvis.

On January 7, 1965 under spinal anesthesia, a laparotomy was performed. Prior to surgery, a long sterile stethoscope was placed in the vagina, and the machinery murmur attested to by three independent observers. The aorta, the bifurcation, the common iliacs, the hypogastrics, the sigmoid colon, and the left ureter were all mobilized with tapes. To the left of the covered cervical stump, attached to it and the urinary bladder was a soft vascular mass, the size of an orange. A continuous bruit was palpated here. First the left common iliac was clamped. The fistula was monitored by a physician with the indwelling stethoscope. This maneuver caused an instantaneous reduction in the fistula at the table and nearly total loss of the machinery murmur. With clamping of the right common iliac all sounds disappeared and the mass vanished. These same observations were repeated clamping only both hypogastric arteries. The left hypogastric artery was then transected and doubly ligated. The left hypogastric was also ligated distal to the take off of the superior gluteal artery. The right hypogastric artery was ligated in continuity with black silk.

Postoperatively the patient had a benign course except for bladder irritation which subsided in five days. She was discharged to follow up.

On March 14, 1965 she was readmitted with an acute abdomen. Laparotomy disclosed volvulus of the jejunum six inches from the ligament of Treitz. Three feet of small bowel were resected for gangrene. Exploration of the pelvis revealed the fistula to be closed. She made an uneventful recovery. The patient is asymptomatic now.

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For approximately three months she experienced left gluteal claudication following strenuous walking.

Discussion

The ideal treatment of the arteriovenous fistula is total interruption of the fistulous tract, with preservation of the arterial and venous limbs. This can usually be accomplished in traumatic fistulas of the neck and extremities and the abdomen. The treatment of congenital fistulas is complicated by the fact that the arteriovenous communications are usually multiple. In most instances total correction of congenital fistulas by surgical measure is nearly impossible.

In the case presented we cannot define with absolute certainty the etiology of the fistula. The procedure used is a practical and safe one. Ischemia of the bladder is rarely seen following bilateral hypogastric artery ligation. It is commonly used in urological surgery, prior to difficult prostatectomy. It is problematical whether total thrombosis of the fistula will occur before collateral circulation can reestablish itself. Technically total interruption of the fistula in this case appeared to present an unwarranted risk.

Summary

An unusual case of a pelvic arteriovenous fistula is presented. The etiology is uncertain. Opportunities existed from numerous D & C's and a low caesarian section. Perforation of a uterus during a D & C is not uncommon, and a low flap section conceivably could injure neighboring vessels. However, the presence of a small atrial septal defect suggest a congenital origin. The management is discussed.

12 Spruce Street, Augusta, Maine

Intraperitoneal Versus Lesser Peritoneal Sac Air in Rupture of a Hollow Viscus

The Falciform Ligament Sign

Demitrios A. Nikolaidis, M.D.*

The purpose of this paper is to consider and emphasize the possibility of early and accurate diagnosis of perforation even in unsuspected cases. It attempts to deal not only with the x-ray detection of the perforation of the abdomen on plain film but also with the precise localization of the free air, thus establishing the site of perforation. The characteristic pattern of air outside the G.I. tract and the visualization of the falciform ligament are the diagnostic signs. These can be present on the plain film of the abdomen or on films of the gallbladder taken for the patient's work up when the perforation might not have been suspected.

Anatomy

The anatomy of the main peritoneal cavity and that of the lesser sac are well known. They communicate through the foramen of Winslow. The round ligament or ligamentum teres represents the obliterated umbilical vein. It extends from the umbilicus in an upward direction to join the left branch of the portal vein. The peritoneum from the anterior abdominal wall reflects around the round ligament. This reflection of the peritoneum forms a somewhat angular fold, the falciform ligament of the liver, which attaches the upper and

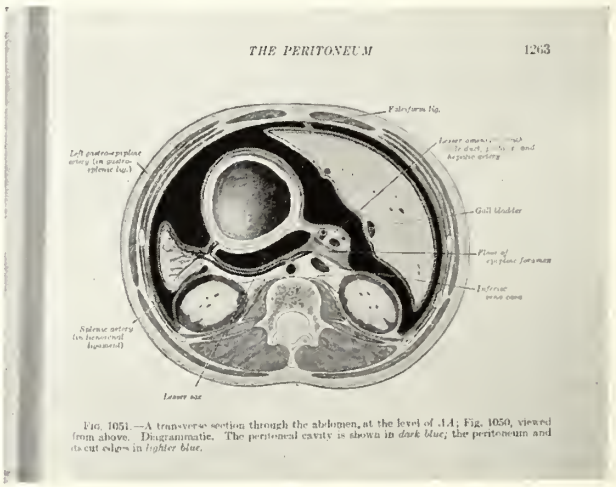


Fig. 1051.—A transverse section through the abdomen, at the level of AA; Fig. 1050, viewed from above. Diagrammatic. The peritoneal cavity is shown in *dark blue*; the peritoneum and its cut edges in *lighter blue*.

Fig. 1. From Gray's Anatomy.

anterior surface of the liver to the diaphragm and abdominal wall.

As can be seen in Fig. 1, the falciform ligament and the right border of the lesser sac lie in the same sagittal plane.

Signs of Perforation

In a case of suspected perforation the most valuable

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Fig. 2, Case No. 1. Note the crescent-shaped radiance.



Fig. 3, Case No. 1. The sight of perforation and the extravasation of the opaque media in the lesser sac are obvious.



Fig. 4, Case No. 2. The curvilinear density at the R.U.Q. is the falciform ligament.

films are the upright and the right lateral decubitus. The free air is seen under the diaphragm or between the liver and the right lateral abdominal wall respectively. The film of the abdomen on the other hand is taken mostly for evaluation of obstruction of the G.I. tract, obliteration of the preperitoneal fat line in a case of peritoneal irritation, soft tissue masses, abnormal calcifications, radiopaque stones. Several diagnostic signs have been described, however, as seen on an abdominal film in a case of pneumoperitoneum, like the bowel wall sign and the football sign. The falciform ligament sign seen on the other hand has not been given very much attention or even looked for. It signifies the presence of free air in the peritoneal cavity.

If the perforation is on the posterior wall of the stomach or duodenal cap and a large amount of air escapes, the entire lesser sac is outlined with its characteristic shape. The stomach may become displaced to the left. It is on the presence of a small amount of air that the diagnosis is difficult and our first case is a representative one.

Banner only in 1945 called attention to a right paravertebral gas shadow. He described it as found in the posterior perforation and in any form of pneumoperitoneum.

Morrison in 1945 reported the signs of free effusion into the lesser sac. He did not, however, discuss air shadows but soft tissue shadows due to extravasation of gastric fluid.

Mellins, indeed, contributed very much with his work and publications in this field.

Case Reports

Case No. 1, Figs. 2 and 3. A 70-year-old white female, had several admissions to the A.G.H. She was known to have a hiatus hernia and an ulcer crater on the lesser curvature of the stomach near the hiatus. The last G.I. showed evidence of healing of the ulcer. She was admitted on 2/23/66 because of epigastric and RUQ pain. Physical examination was essentially negative. Flat plate of the abdomen and upright films were unremarkable.

The next day she complained of Rt shoulder pain. On 2/28/66 she had a G.B. series. There was no evidence of chololithiasis. A peculiar shaped radiolucency was seen in the RUQ on the film (Fig. 2). The nature of this could not be determined at that time. A G.I. series done on 3/1/66 showed the site of perforation and extravasation of the opaque material in the lesser peritoneal sac (Fig. 3). She was managed medically, did well and was discharged.

The peculiar air shadow on the film of the G.B. represented air outlining the right contour of the lesser peritoneal sac.

Case No. 2, Fig. 4. This 44-year-old white female was admitted on 4/11/66 because of upper abdominal pain. The pain was in the RUQ with radiation to her back and had started 24-hours prior to her admission. She was nauseated and vomited at home.

Physical examination was essentially negative except for pain and some tenderness at the RUQ. The clinical impression was cholecystitis. The next day she came to the x-ray department for a G.B. series. On the preliminary film of the abdomen, however, Fig. 4, the diagnosis of perforation of a hollow viscus was made. The large amount of free peritoneal air outlined the falciform ligament on both sides. In view of this the patient was sent to the operating room and an anterior gastric perforation was found. She had an uneventful recovery and days later she was discharged.

Summary

The curvilinear density seen at the R.U.Q. represents the falciform ligament. This is only present when there is air on both sides of the ligament and it means perforation of a hollow viscus.

On the other hand the crescent-shaped radiance seen at almost the same plane on the R.U.Q. represents air in the lesser peritoneal sac outlining its right border. It means posterior gastric or duodenal cap perforation.

Mellins states that small loculated collections of gas in the lesser peritoneal sac are difficult to be identified without lateral views of the abdomen after administration of contrast media. Our first case, however, accidentally proved that by raising the patient's right side up the small amount of air tends to accumulate under the right side of the lesser sac wall and becomes visible.

The very recent experience with the two consecutive cases made us feel that these signs can help establish the diagnosis of a perforated viscus where neither the history nor the findings on the physical examination are of definite value to the clinician.

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Computer at the Bedside?

As to the evaluation of medicines for efficacy and safety, the computer is not the final and perfect answer, useful though it is. What the physician feels and perceives at the bedside of his patient may not fit into the square, or oblong or round hole of the punch card; but his observations are often a surer guide to the usefulness of a particular medicament for a particular patient. In the interest of the patient – that individual so unique that there is not another entirely like him in the whole wide world – we must be careful lest the scientific pendulum swing too far in the direction of mechanistic technology. – J. Mark Hiebert, M.D., to University of Kansas Pharmacy Colloquium, April 13, 1966.

Amniotic Fluid Analysis To Improve Fetal Survival

GEORGE R. LANDWEHR, M.D. and LEE W. RICHARDS, M.D.*

The purpose of this paper is to report a case of an Rh sensitized patient and the use of amniotic fluid analysis to determine the optimum time for delivery.

CASE REPORT

This patient is a 26-year-old, Gravida VI, Para IV, type O, Rh negative, white female. First pregnancy, 1959, 6 pound 15 ounce female at term; second pregnancy 1961, a female at term, had two exchange transfusions; third pregnancy 1962, male 34½ wks. gestation, infant died in the neonatal period with hydrops fetalis; fourth pregnancy in 1964, a male at term, had three exchange transfusions; fifth pregnancy in 1965 ended in a 3 mos. spontaneous abortion. With this present pregnancy the last menstrual period was April, 1965. The exact date was unknown by the patient. This patient was first seen in our Clinic November 23, 1965. She lived out of town but had no prenatal care prior to this. On her first appearance at this Clinic a blood anti-titer was taken and it was positive in the undiluted saline, negative diluted. It was positive to 1:64 albumin. The approximate size of the fetus by clinical examination was 6 mos. A second blood specimen taken December 15, 1965 was positive in the undiluted saline, negative in the diluted saline. It was positive 1:16 in the albumin fraction. Another specimen taken on December 28 (two weeks later) showed negative titer in the saline and positive in 1:16 albumin. This showed that there was an apparent drop in the titer. Whether or not the previous titers prior to her admission were very high and had dropped to the level first seen at this Clinic is unknown. In contrast to the blood analyses amniocentesis was performed for the first time December 1, 1965. This revealed an abnormal curve (Fig. 1). This was high enough to be in the area which would need an intrauterine transfusion or delivery of the baby depending upon the time of gestation. A second amnicentesis done two weeks later (12-14-65) showed some improvement (Fig. 2). A third amniocentesis performed one week later again showed improvement (Fig. 3). The last amniocentesis done on January 11, 1966 was even more improved (Fig. 4). Because of these improving amniocentesis analyses the patient was allowed to go to term and was induced with I.V. Syntocinon® drip and amniotomy. She had an uneventful labor, delivered an apparently normal 5 pound 7 ounce male infant spontaneously. Cord blood was taken and revealed an elevated bilirubin and a positive Coombs. The infant received three transfusions and was discharged in good condition.

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DISCUSSION

This patient was not seen in our Clinic until the 6th month of gestation. Blood anti-titer test showed a positive reaction that was not very high. We did not know whether this level reflected a precipitous drop from a previously high level or this was her initial level. Sub-



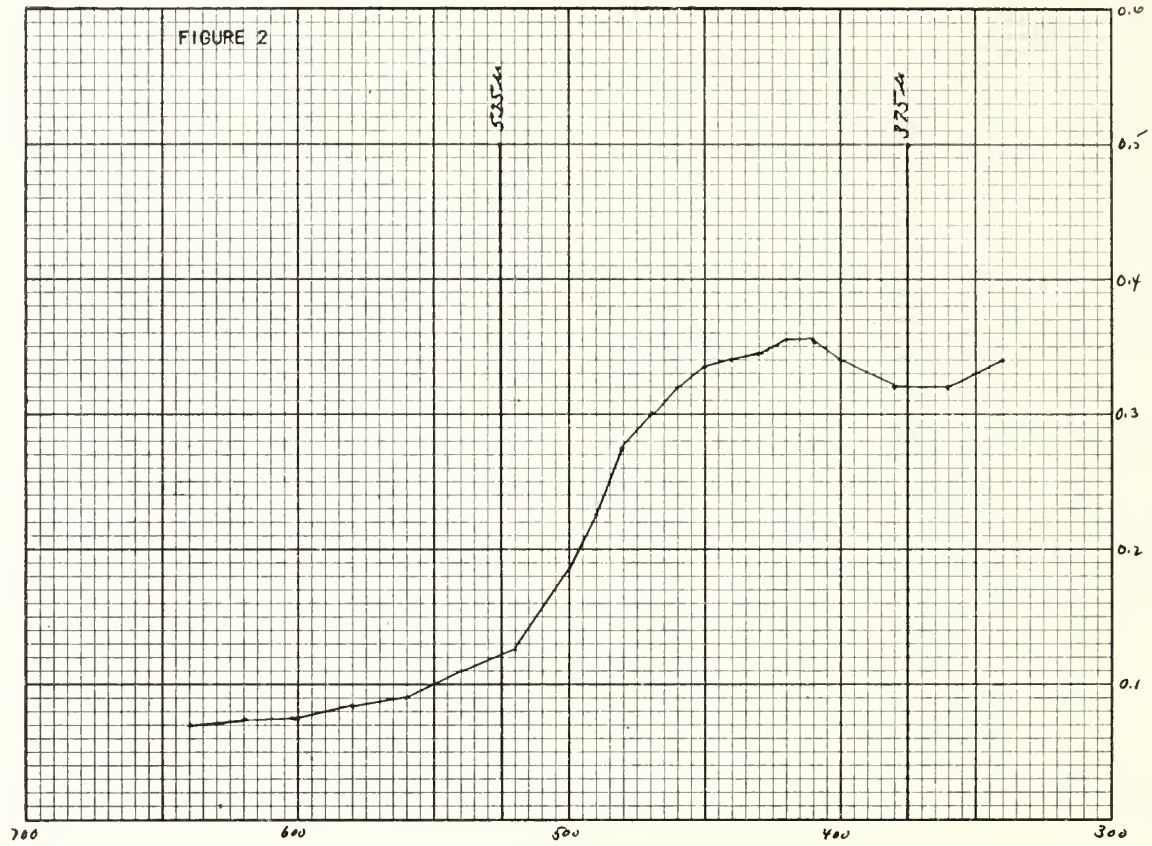
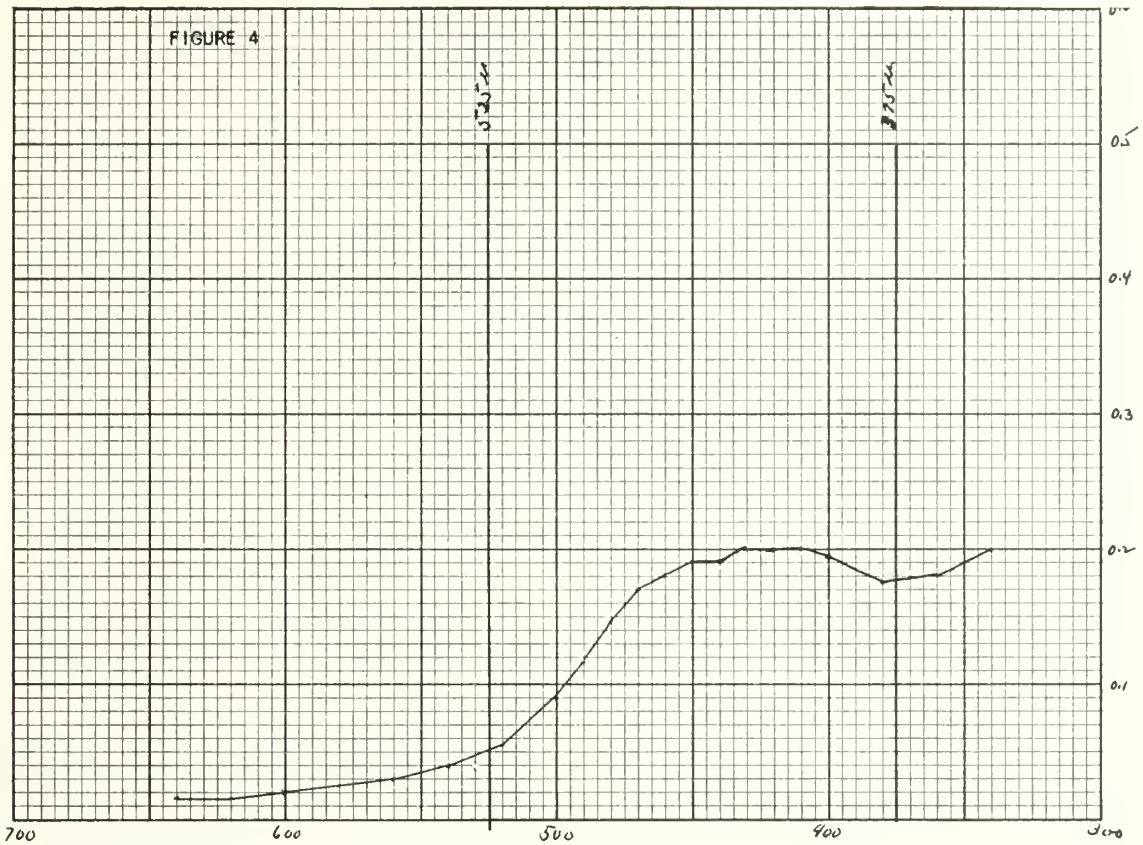

FIGURE 2
325mμ
375mμ
0.0
0.5
0.4
0.3
0.2
0.1
700
600
500
400
300


FIGURE 3
325mμ
375mμ
0.5
0.4
0.3
0.2
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700
600
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sequent readings were even lower and showed some tendency to drop. Based on the past history and the possibility of a rapidly falling titer early delivery might be decided even though the fetus was less than 34 wks. gestation. This, however, would further jeopardize a compromised infant with the even greater problem of prematurity. There are times when an anamnestic reaction is found post delivery when an Rh negative infant is delivered and the mother had had a rising titer.

The amniocentesis determinations determine the bilirubin pigment present in the amniotic fluid as a result of breakdown of fetal blood cells. In this case even though the initial tap was elevated to a dangerous level the subsequent tests were improved and allowed the fetus to go to term. The exact mechanism of the elevated tap the first time which subsequently returned to normal is unknown.

Conclusion

Perinatal mortality has declined in the past decade due to early delivery and exchange transfusions based on the anti-titer of the mother's blood. At best this test is somewhat inaccurate and does not necessarily relate to the physician the optimum time for delivery. We have all seen positive titers in Rh negative babies. Many times a low titer that is not rising rapidly may delude the physician into thinking there is no need for early delivery. On occasion the fetus is delivered prematurely, compounding the problem.

Spectrophotometric analyses of the amniotic fluid after amniocentesis gives a very accurate estimation as to the status of the fetus. One determination is of little value. Serial determinations must be performed. This test does seem to be the most accurate measurement to determine the optimum time for delivery and also, where possible, early intrauterine transfusions in order to allow the fetus to remain in utero until the time of delivery without being compromised. This test shows not only when to deliver but, just as important, when not to deliver as is illustrated in the case presented.

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Dr. Richards, 21 Western Avenue, Augusta, Maine

The Don'ts of Fracture Care

JOSEPH R. CRAWFORD, M.D., F.A.C.S.*

Having read numerous monographs on the care and treatment of fractures, sprains, and dislocations, I couldn't recall any dealing specifically with the pitfalls to be avoided in the treatment of skeletal injuries. Certainly nothing included here is new by any stretch of the imagination, however, I felt it might be of some use to list possible pitfalls of fracture care as I see them.

1. Don't assume that all fractures can be diagnosed clinically. Many fractures would go unrecognized without x-ray's. Missing a fracture may lead to converting an undisplaced fracture into a displaced fracture. The resultant of this is unnecessary injury to a patient and possibly fertile ground for legal action.

2. Don't fail to assess the nerve and arterial status of the extremity at the time of examination. If nerve or arterial impairment is present be sure and notify the patient or his parents prior to manipulation or treatment.

3. Don't fail to notify parents of patients with epiphyseal injuries of the possibility of growth disturbances. This may be a little shocking to the parent; however, when presented properly in terms of the low incidence of occurrence, it will be appreciated, and you are protected.

4. Don't fail to point out to a patient treated for a fracture what activity is, and what is not permissible on returning home. Inform him that if he has undue pain, swelling, or discoloration of the toes or fingers to notify you immediately.

5. Don't fail to notify fracture patients of the time of each return visit.

6. Don't fail to immobilize the joint above and below all displaced fractures requiring manipulation.

7. Don't fail to adequately pad casts, especially about bony protuberances.

8. Don't fail to obtain recheck x-ray's on all fractures to determine if the position is being maintained. X-ray at weekly intervals for relatively unstable fractures until adhesion has occurred.

9. Don't fail to listen to a patient complaining of severe pain in plaster. Immediate careful examination is called for, and if in doubt bivalve or remove the cast immediately.

10. Don't hesitate to correct displacement of fractures occurring in plaster if the position of the fragments becomes other than satisfactory. This may require wedging of the plaster, or complete remanipulation under anesthesia, or even open reduction. A surgeon must be equipped to go all the way, and cannot stop short of satisfactory alignment of the fragments.

11. Don't rush to open fractures. Many fractures which at first on x-ray appear destined for operative reduction may be reduced by conservative measures.

12. Don't delay unduly to open fractures which do not respond to one or two attempts of experienced closed manipulation.

13. Don't expect to obtain good closed reductions on badly displaced fractures associated with severe swelling of the extremity. Permit the swelling of the extremity to diminish prior to manipulation, if the circulation is adequate. Even a week is not too long to wait.

14. Don't cultivate the philosophy of "getting away with it". The position of fracture fragments is either acceptable, or not acceptable. We must rely on x-ray for this judgment.

15. Don't fail to remember that large amounts of blood may pour into the tissues with certain fractures, especially of the femur, and shock may occur rapidly. Adequate measures must be taken promptly.

16. Don't fail to use tetanus toxoid, or antitoxin and antibiotics in the treatment of open fractures.

17. Don't fail to adequately examine every fracture patient prior to treatment, and obtain a complete history as to allergies, etc. Remember associated injuries, for example ruptured spleen, kidneys, etc., are not rare in this group of patients.

18. Don't fail to use bone grafts in association with open reductions requiring plates and nails, with the exception of hip fractures.

19. Don't fail to maintain adequate immobilization of all fractures until there is x-ray evidence of union. If a cast becomes loose with atrophy of the arm or leg, or a decrease of swelling, the cast should be changed.

20. Don't fail to recognize your own limitations, and have adequate and prompt consultation on any fracture you do not feel completely confident in handling.

21. Don't fail to recognize early signs of embolism, and be prepared to take the necessary steps, whether it be anticoagulation or vein ligation.

22. Don't fail to follow fracture cases with adequate exercises and physiotherapy following removal of the cast. Remember if you don't follow your fracture cases carefully they will follow you.

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Modified Anterior Tibial Compartment Syndrome

Allan J. Stinchfield, M.D.

One of the rare causes of pain in the leg is the anterior tibial syndrome consisting of symptoms and changes caused by compression and ischemia. This syndrome was first described by Vogt, in 1943, as an acute ischemia or necrosis of the musculature occurring without trauma. In 1948, Hughes reported on "The Necrosis of the Anterior Tibial Muscles Due to Fatigue." In 1956, Mavor et al, gave a case report of a patient who had severe anterior leg pain on exercise. The symptoms were relieved by linear sectioning of the deep fascia.

Today, the highest incidence of this condition is found in the Armed Services. Young recruits are given strenuous leg exercise. At the end of a long march, they may suddenly develop severe pain, with or without swelling, in the anterior lateral aspect of the leg. These symptoms, due to compression and ischemia within the tibial compartment, are usually relieved by rest and elevation. If the pain is persistent and intractable, surgical intervention is advisable before definite necrosis of the musculature occurs.

The anterior tibial compartment is bounded; medially, by the lateral surface of the tibia, the interosseous membrane and the lateral surface of the fibula; posteriorly and laterally, by the deep fascia of the leg. This compartment, which extends from the knee to the ankle, contains the following: the anterior tibial muscle; the long extensors of the toes; the extensors hallucis, proprius, and longus; the deep and superficial peroneal nerves; the anterior tibial artery and vein; the peroneal muscles, longus, brevis and tertius. The transverse crurial ligament, at the anterior ankle joint, extends from the tibia to the fibula and overlies the tendons of the above mentioned muscles.

Case Report

The following case is presented because it was somewhat more chronic than most of those reported and had additional neurological symptoms. Mr. S., age 49, fractured his left leg in 1959. The bones healed readily but, since then, when walking, he had sharp pain in the anterior part of the leg. Onset of pain was after walking one block. At times, he had swelling in the distal leg. The pain occasionally disturbed his sleep. During the last six years, sometimes, he took as many 25 aspirin a day without relief. His other leg had given him similar but less severe discomfort.

Analysis of the pain revealed that, when he was standing or walking, it started at a point lateral to the tibial crest in the distal third of the leg where a small swelling also would appear. Accompanying the pain was paraesthesia and numbness over the distal anterior leg and foot.

The presumptive preoperative diagnosis was not anterior tibial compartment syndrome but traumatic neuritis due to pinching of the superficial peroneal nerve where it leaves the deep compartment distally. It was thought that the nerve was squeezed by the underlying muscle at the tight rim of the aperture in the deep fascia.

In November of 1965, some six years after the onset of the symptoms, the left leg was operated upon. The patient had so much relief that he returned a month later and requested an operation on the other leg.

The operations were nearly identical. A five inch incision was made lateral to the tibial crest in the distal third of the leg. Subcutaneous fat and skin were reflected from the deep fascia for a half-inch on either side of the incision in an effort to find the aperture in the deep fascia where the superficial peroneal nerve becomes subcutaneous and branches to become the medial and intermediate dorsal cutaneous nerves of the foot. No nerves were found in the superficial fascia but they could be seen through the deep fascia. The overlying deep fascia was then incised directly over the nerves from a point about 1″ proximal to the ankle, to the mid-portion of the leg. There were no signs of tumor or constrictive rings about the nerves. Their color was good. There were no signs of impaired circulation in the muscles or nerves. In the left leg, the fat surrounding the medial cutaneous nerve was somewhat more yellow than the fat in the neighboring area. The significance of this discoloration is not known.

Following the surgery, the patient was almost immediately free of pain and paraesthesia. Soon he could walk long distances without pain and swelling.

It was postulated that this was an anterior compartment syndrome which, besides compression and ischemia of the mucles, gave pressure symptoms on the superficial peroneal nerves that resulted in pain and anaesthesia of the leg and foot.

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State Of Maine

Department of Health and Welfare

DEAN H. FISHER, M.D.
COMMISSIONER

Ambulance and Emergency Department Services in Maine

PAUL F. PAETOW*

The Department of Health and Welfare staff have completed a report describing Ambulance and Emergency Department Services in Maine. This report should be of interest to Maine physicians, who are in daily contact with these services; especially those physicians interested in improving services in their communities.

As a result of statewide interviews with almost all ambulance operators and with persons associated with emergency departments of hospitals, the following facts were found:

The populated portions of the State are well covered by ambulance services – 75% of which are operated by funeral directors.

Seventeen percent of the calls are the results of motor vehicle accidents (many involving more than one person), 11% are for home and industrial accidents and 25% are for non-accident emergencies. The rest are local and long-distance transfers of non-emergency patients.

About one-third of the ambulance respondents stated that physicians, (both M.D. and D.O.) go to the scene of accidents. In many cases, however, first aid at the scene is rendered by police and ambulance personnel.

Only a small proportion of ambulance personnel have received any formal training in first aid. The first aid policy of most units is that of "minimal life saving – i.e. restore breathing and control hemorrhage – with little or no splinting or emphasis on shock. Apparently wounds are rarely bandaged unless bleeding is profuse or life threatening.

Communications from the scene of an accident to the hospital are limited and indirect. Limited, because a minority of units (i.e. approximately one-third of the funeral services) have radios. (They must rely on police or other citizens to contact the hospital.) Indirect, because even those units having radios must call their headquarters, which in turn relay the message to the hospital by telephone.

Even where the ambulances or police contact the hospitals, the nurse in charge must frequently evaluate the patient herself before calling a physician.

A large variety of emergency department rotating, "on call" schedules for physicians exist. Many of these are of doubtful effectiveness – e.g. where a physician is on call for a month at a time.

Emergency department personnel generally see little evidence of first aid having been rendered to patients brought in by ambulance.

Yet, there is general satisfaction among hospital staff and personnel with the condition of ambulance patients.

The report contains thirteen recommendations for improving emergency care of the sick and injured. Many of these are directed toward the Maine Medical Association and its Committee on Trauma. Among these recommendations are:

A review of the desirability of having physicians go to the scene of an accident to give first aid.

If this is found undesirable, general policies regarding the kinds of and quantity of first aid to be rendered at the scene – by ambulance and rescue personnel – ought to be formulated.

Policies setting forth the training required, to prepare personnel to give this care at the scene of an accident, should be developed by the Maine Medical Association or its Committee on Trauma.

Local hospital-centered, physician-led emergency ambulance training courses such as the well known Knox County EAS Course should be established in other Maine communities.

Rotating "on-call" systems by hospital staff and administrations for maximum assurance that the "on-call" physician can be available for the entire period of his duty should be established. It should also be routine for the charge nurse to alert the "on-call" physician when she hears that a serious case is on the way to the hospital.

Other recommendations relate to direct communications systems (ambulance-hospital), establishment of volunteer rescue and first aid units in rural and suburban areas, legal protection for ambulance personnel rendering first aid and the question of the desirability of licensing statutes.

The report contains 100 pages of text and supporting material, in the form of tables summarizing the data.

The report is a public document and the Department wishes to provide a copy (or an opportunity to read the report) to interested physicians and laymen, who can make use of the information.

Requests for copies will be honored. Inquiries about the report will be welcomed by the Accident Prevention Program of the Department.

*Accident Prevention Director

In Diverticulitis...

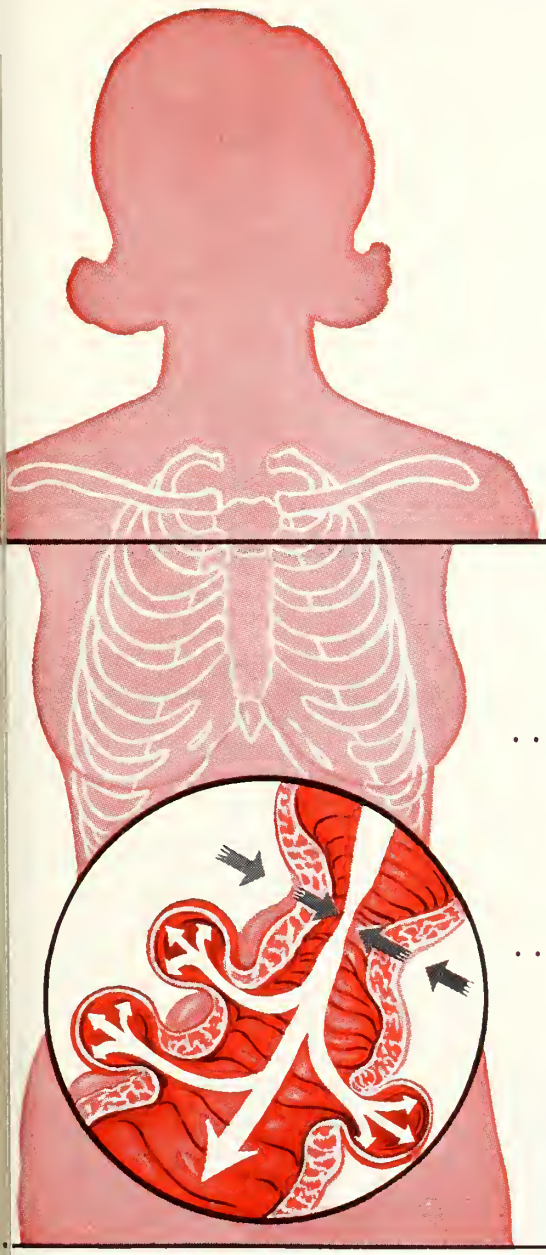
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SEARLE

Research in the Service of Medicine

From the Secretary's Notebook

113th Annual Session of the M.M.A. House of Delegates

Sixty-three delegates and alternates and nineteen guests registered for the 113th annual session at The Samoset, Rockland, Maine on Sunday, June 12, 1966. George E. Sullivan, M.D., President-elect of the M.M.A., called to order the meetings of the House, which were presided over by Linus J. Stitham, M.D., Speaker of the House.

Election of Speaker and Vice Speaker of the House of Delegates Meetings to be Held in 1967 — Robinson L. Bidwell, M.D. of Portland was elected Speaker of the House of Delegates and Charles R. Glassmire, M.D., Vice Speaker of the House.

Budget for 1967 and Increase in Dues — Copy of the proposed budget for fiscal year 1967 as presented at the Interim Meeting of the House of Delegates by Edward K. Morse, M.D., Chairman of the Budget Committee, was sent to each delegate and alternate following the Interim Meeting for study before final action. The proposed budget for 1967 was approved as was the proposed increase in 1967 dues from $55.00 to $65.00 annually.

Nominating Committee — The report of the Nominating Committee consisting of nominees for Standing Committees for 1966-1967 and for a delegate and alternate delegate to the AMA to serve from January 1, 1967 to January 1, 1969 was accepted. This report was published in the July, 1966 issue of The Journal of the Maine Medical Association, page 140.

Report of Ad Hoc Committee on Osteopathy — Copy of this report was sent to all delegates and alternates following the Interim Meeting in April. The following motions were approved at the annual session:

That it is not unethical to participate in or sponsor scientific educational functions to which individual Doctors of Osteopathy are invited.

That it is not unethical for a hospital staff to extend to individual Doctors of Osteopathy such access to ancillary and consultative hospital services as the staff and credentials committee deem necessary for the welfare of the patients in its area.

That the Ad Hoc Committee be dissolved and that a special committee of the Maine Medical Association be created to deal with interprofessional relations.

Public Relations Committee — A proposal that each candidate for membership on the Public Relations Committee must be a duly elected member of the Council at the beginning of his term of office on the committee was rejected, and it was voted that the makeup of the committee remain "as is."

It was voted that the function of the M.M.A. Public Relations Committee and its relationship to the Public Relations Committee of the county societies be referred to the Council for study and report back to the House of Delegates at its next Interim Meeting.

Board of Ethics and Discipline — It was voted that the President appoint a special committee to study, with appropriate legal advice, the development of the Board of Ethics and Discipline based on the State of Washington's example, as proposed by Harry Brinkman, M.D., Chairman of the Board.

Legislative Committee — The House of Delegates voted in opposition to S2568, the so-called Medical Restraint of Trade Act, which has to do with prohibition of kick-backs in dispensing drugs or other devices. It also voted in opposition to HR12976 "The Adult Medical Protection Act of 1966."

Resolution Submitted by Committee on Problems of Long-Term Patient Care and Treatment — This resolution, which follows, was approved.

As Chairman of the Committee on Problems of Long-Term Patient Care and Treatment, I would like to submit the following draft for a Resolution to the House of Delegates:

Whereas, there exists a dire need of beds for chronic care patients requiring hospitalization for from 6 to 12 months, and even more so for patients who will need modified protective care services for a lifetime: Therefore, be it Resolved that the Maine Medical Association go on record to urge the Governor of the State of Maine and the members of the 103rd Legislature, as well as the Health Facilities Planning Council of the State of Maine, the Department of Mental Health and Corrections, the Department of Health and Welfare, and the Boards of Trustees of Maine's general hospitals, to actively engage in a regional planning of facilities for the above-named groups of patients, as soon as possible.

If there is a desire to modify this Resolution in any way, this will, of course, be up to the House of Delegates.

Peter W. Bowman, M.D., Chairman

Committee on Liaison Activities Between the Maine State Nurses' Association and the M.M.A.— A proposal by this committee that if nurses are properly trained they should be allowed to initiate closed chest Cardio-Pulmonary Resuscitation, as an emergency measure until a physician arrives, was approved.

The proposal relative to nurses reporting to the Executive Director of the M.M.A. was rejected as was a proposal concerning nurse education.

Election of Councilors — Charles R. Glassmire, M.D. of Portland was elected Councilor for the First

Continued on Page 182

Elected at the 113th Annual Session of the Maine Medical Association

Rockland, Maine June 12, 13, 14, 1966

President-Elect

PAUL S. HILL, JR., M.D., Saco

Councilor, First District

CHARLES R. GLASSMIRE, M.D., Portland

Councilor, Second District

CHARLES F. BRANCH, M.D., Lewiston

Council Chairman

ASA C. ADAMS, M.D., Orono



DR. HILL



DR. GLASSMIRE



DR. BRANCH

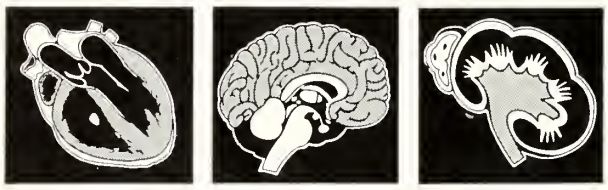


DR. ADAMS

Behind continued high blood pressure readings lies the possibility of organic damage[1-13]

MANY OF THE aspects of essential hypertension are unpredictable—either because there are a number of mechanisms involved or because individuals differ in their responses to these mechanisms.[1]

There is one aspect of hypertension, however, that seems, in many cases, predictable. ". . . when the blood pressure is elevated to a marked degree for an adequate period of time, this in itself leads to perpetuation of the syndrome with resulting vascular damage throughout the body."[14] All too often the disease progresses until there is damage to one of three vital organs: the heart, the kidney, the brain.



"Hypertension is certainly a major factor in the genesis of coronary heart disease, and it is even more important when compounded with obesity."[4]

"[Vascular deterioration] can be clearly seen in the kidney with a degree of damage that can be measured by renal function studies."[10]

". . . most evidence suggests that reduction of blood pressure, when it is too high, not only relieves the heart of excess work but reduces vascular damage."[1]

"In short, treatment is indicated."[1]

Antihypertensive therapy will not restore the blood vessels to normal. Yet many of the vascular changes and symptoms caused by increased blood pressure may be arrested or alleviated when the blood pressure is reduced to normotensive levels.[7]

Reducing the blood pressure helps curtail further vascular damage and improves the prognosis — when damage is not too far advanced before therapy is started.[14] Essential hypertension is an indication not only for treatment, but for *early* and adequate treatment of the patient in question.

Reduce the blood pressure with Rautrax-N

Rautrax-N combines the antihypertensive-tranquilizing action of whole root rauwolfia with the antihypertensive-diuretic action of bendroflumethiazide in one convenient medication. The two drugs complement each other so that smaller doses of both are possible.

Rauwolfia combined with bendroflumethiazide is particularly effective in long-term therapy,[15-17] since beneficial effects do not diminish with continuous daily administration.

For most patients 1 or 2 Rautrax-N tablets daily are sufficient for maintenance therapy. The simplicity, convenience and economy of such a dosage schedule are of particular benefit to older patients.

References: 1. Page, I. H., and Dustan, H. P.: The Usefulness of Drugs in the Treatment of Hypertension, in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 95. **2.** Hollander, W.: The Evaluation of Antihypertensive Therapy of Essential Hypertension in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 97. **3.** Nickerson, M.: Antihypertensive Agents and the Drug Therapy of Hypertension, in Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 3, New York, The Macmillan Co., 1965, p. 727. **4.** Berkson, D. M.: Indust. Med. & Surg. **32**:371, 1963. **5.** Cohen, B. M.: M. Times **91**:645, 1963. **6.** Lee, R. E., et al.: Am. J. Cardiol. **11**:738, 1963. **7.** Moyer, J. H.: Am. J. Cardiol. **9**:821, 1962. **8.** Moser, M.: New York J. Med. **62**:1177, 1962. **9.** Wood, J. E., and Battey, L. L.: Am. J. Cardiol. **9**:675, 1962. **10.** Moyer, J. H., and Heider, C.: Am. J. Cardiol. **9**:920, 1962. **11.** Moser, M., and Macaulay, A. I.: New York State J. Med. **60**:2679, 1960. **12.** Judson, W. E.: Nebraska M. J. **44**:305, 1959. **13.** Hodge, J. V.; McQueen, E. G., and Smirk, H.: Brit. M. J. **1**:5218, 1961. **14.** Moyer, J. H., and Brest, A. N.: Hypertension Recent Advances, Philadelphia, Lea & Febiger, 1961, p. 633. **15.** Berry, R. L., and Bray, H. P.: J. Am. Geriatrics Soc. **10**:516, 1962. **16.** Reid, W. J.: J. Am. Geriatrics Soc. **13**:365, 1965. **17.** Feldman, L. H.: North Carolina M. J. **23**:248, 1962.

Contraindications: Severe renal impairment or previous hypersensitivity. **Warning:** Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting or G.I. bleeding occur.

Precautions and Side Effects: The dose of ganglionic blocking agents, veratrum or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Caution is indicated in patients with depression, suicidal tendencies, peptic ulcer; electrolyte disturbances are possible in cirrhotic or digitalized patients. Marked hypotension during surgery is possible; consider discontinuing two weeks prior to elective surgery and observe patients closely during emergency surgery. Rauwolfia preparations may cause reversible extrapyramidal symptoms and emotional depression, diarrhea, weight gain, edema, drowsiness may occur. Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients, and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, rashes may occur.

Dosage and Supply: Initial dosage, 1 to 4 tablets daily, preferably at mealtime. Maintenance, 1 or 2 tablets daily. Rautrax-N is supplied as capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin®), 4 mg. bendroflumethiazide (Naturetin®), 400 mg. potassium chloride.

Also available: Rautrax-N Modified — capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin), 2 mg. bendroflumethiazide (Naturetin), 400 mg. potassium chloride. Both potencies available in bottles of 100. For full information, see Product Brief.

RAUTRAX®-N

Squibb Rauwolfia Serpentina Whole Root (50 mg.) with Bendroflumethiazide (4 mg.) and Potassium Chloride (400 mg.)

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'The Priceless Ingredient' of every product is the honor and integrity of its maker.

Maine Medical Association

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Special Committees for 1966–1967 as appointed by the President, George E. Sullivan, M.D. of Fairfield.

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Hamdi Akar, M.D., 17 Grove St., Bath
John P. Dow, M.D., 59 Main St., Pittsfield
Elton R. Blaisdell, M.D., 12 Deering St., Portland

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Harold A. Pooler, M.D., Bangor State Hospital, Bangor
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Henry C. Thacher, M.D., 117 Goff St., Auburn

Committee On Rehabilitation

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John A. Woodcock, M.D., 35 Second St., Bangor
Edward G. Asherman, M.D., 131 Chadwick St., Portland

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Milan A. Chapin, M.D., 237 Turner St., Auburn
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Eugene C. McCann, M.D., 49 Deering St., Portland
Stanley W. Kent, M.D., 42 Deering St., Portland
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Charles F. Branch, M.D., Central Maine General Hospital, Lewiston

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Merle S. Bacastow, M.D., 22 Bramhall St., Portland

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Niles L. Perkins, Jr., M.D., Box 1489, Portland

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Richard H. Dennis, M.D., 33 College Ave., Waterville
Oakley A. Melendy, M.D., Doctors Park, 89 Hospital St., Augusta

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William E. Schumacher, M.D., 14 Westwood Rd. MD "B", Augusta
Marvin C. Adams, M.D., 25 Bramhall St., Portland
E. Charles Kunkle, M.D., 131 Chadwick St., Portland
Maurice Ross, M.D., 372 Main St., Saco
John F. Dougherty, M.D., 112 Front St., Bath

*Advisory Committee To The Diabetes Detection Program Of The Department Of Health & Welfare

Melvin Bacon, M.D., 122 Main St., Sanford
Harold D. Cross, M.D., Main Rd. & Summer St., Hampden Highlands

*Appointed by the Council on 2/13/66

To be continued

Department of Health and Welfare

Maine Tuberculosis and Health Association

Statement of Current Therapy of Tuberculosis

by

Maine Thoracic Society

The treatment of tuberculosis to be effective must be consistent in application. This means:

1. Isolation of patients who are contagious
2. Chemotherapy
3. Education
4. Lifelong follow-up
5. Surgery when indicated after careful investigation by a specialist in chest diseases and after consultation with a chest surgeon

Chemotherapy of tuberculosis has undergone changes since the advent of anti-tuberculosis drugs and will continue to do so as new drugs become available.

WHO SHOULD RECEIVE DRUGS?

1. All active cases of tuberculosis — pulmonary or extra-pulmonary, irrespective of sputum findings. This includes active primary infections as well as reactivated known tuberculosis (relapses).
2. Patients in whom treatment is directed towards prevention of active disease. These include:
   A. Recent tuberculin skin test converters
   B. "Special risk" patients, such as patients on long term steroid therapy, patients recovering from certain viral infections as measles or whooping cough
   C. Hospital and institutional personnel with known exposure and negative skin tests
   D. Old "inactive" disease in patients with extensive fibrosis and calcifications who have not previously had adequate chemotherapy
3. **Potential tuberculosis** — those individuals whose skin tests are negative but have intense exposure to the disease, as children under the age of 5 years. Chemotherapy for this group is true chemoprophylaxis.

WHAT IS ACTIVE TUBERCULOSIS?

Reasonable certain diagnosis depends on:

1. Positive tuberculin skin test. A doubtful Tine test should always be verified by a Mantoux test with PPD intermediate strength.
2. X-ray evidence of a lesion consistent with tuberculosis (pulmonary or extra-pulmonary) and/or smear and culture demonstrating tubercle bacilli.

   If a patient has an X-ray lesion compatible with tuberculosis and a positive skin test supports this suspicion, chemotherapy should be undertaken even if a positive sputum cannot be obtained.

   Consultation is available without charge to the patient at the following places:

   1. Division of Tuberculosis Control, Department of Health and Welfare
   2. Central Maine Sanatorium, Fairfield
   3. Chest Clinics:

      Maine Medical Center — Portland
      Central Maine General Hospital — Lewiston
      Augusta General Hospital — Augusta
      Bangor-Brewer TB and Health Association — Bangor
      Community General Hospital — Fort Fairfield
      Knox County General Hospital — Rockland

The establishment of more hospital-based clinics in the foreseeable future is anticipated. Physicians will be notified of locations as these clinics become operative.

Sanatorium admission for all infectious patients or those who in all likelihood will require close supervision is advised. Initiation of treatment, isolation, and patient education can be carried out more effectively in a sanatorium. So-called "treatment failures", patients with a history of drug resistance and individuals who are mentally or emotionally incapable of cooperating, should also be urged to enter the sanatorium for more effective treatment management.

Patients who have failed to respond to the so-called "first line" drugs (INH, PAS and Streptomycin) will require careful sputum studies, including drug sensitivity and possible surgical intervention. The less common anti-tuberculous drugs as Pyrazinamide, Seromycin,® Trecator, Viocin® (injectable) and their combinations are more toxic; therefore their use in "home treatment" does not seem suitable at the present time.

WHAT IS ADEQUATE CHEMOTHERAPY?

In active tuberculosis it is mandatory to use combined drug therapy to prevent emergence of resistant bacteria.

ISONIAZID (INH): Supplied as tablets (50 and 100 mgm.) and syrup (10 mgm. per 1 cc.) is usually given 5 mgm./kg. of body weight in divided doses for children. An average adult requires 300 mgm. daily. Much higher doses are being used by some clinicians, but the indication must be determined on an individual basis.

Occasionally isoniazid produces peripheral neuritis for which B6 is recommended. This is given in the form of Pyridoxine 25 mgm. T.I.D. or Q.I.D. when indicated.

STREPTOMYCIN: Streptomycin sulphate is given by intramuscular injection only. 1 gm. daily during the acute stage of the disease may then be reduced to 1 gm. 2 times a week.

PARA-AMINOSALICYLIC ACID (PAS): Available as sodium or potassium salt given orally 12 gm. daily in divided doses, usually after meals. Because of frequent gastric intolerance, it may be given with antacids. It is available in tablet form or packets of powder soluble in water.

A combination of INH and PAS is the most commonly used and has the advantage of oral administration. However, if for some reason the above combination is not advisable, PAS may be substituted by Streptomycin (INH plus Strep.) or INH may be substituted by Streptomycin (Strep. plus PAS). Often all three drugs are required to begin treatment and later reduced to two drugs.

DURATION OF CHEMOTHERAPY

Once a diagnosis of active tuberculosis has been established definitely, combined chemotherapy should be started at once and should **NOT BE INTERRUPTED** but continued for a minimum of two years after the disease has stabilized. Once chemotherapy has been started it is more effective when given uninterruptedly.

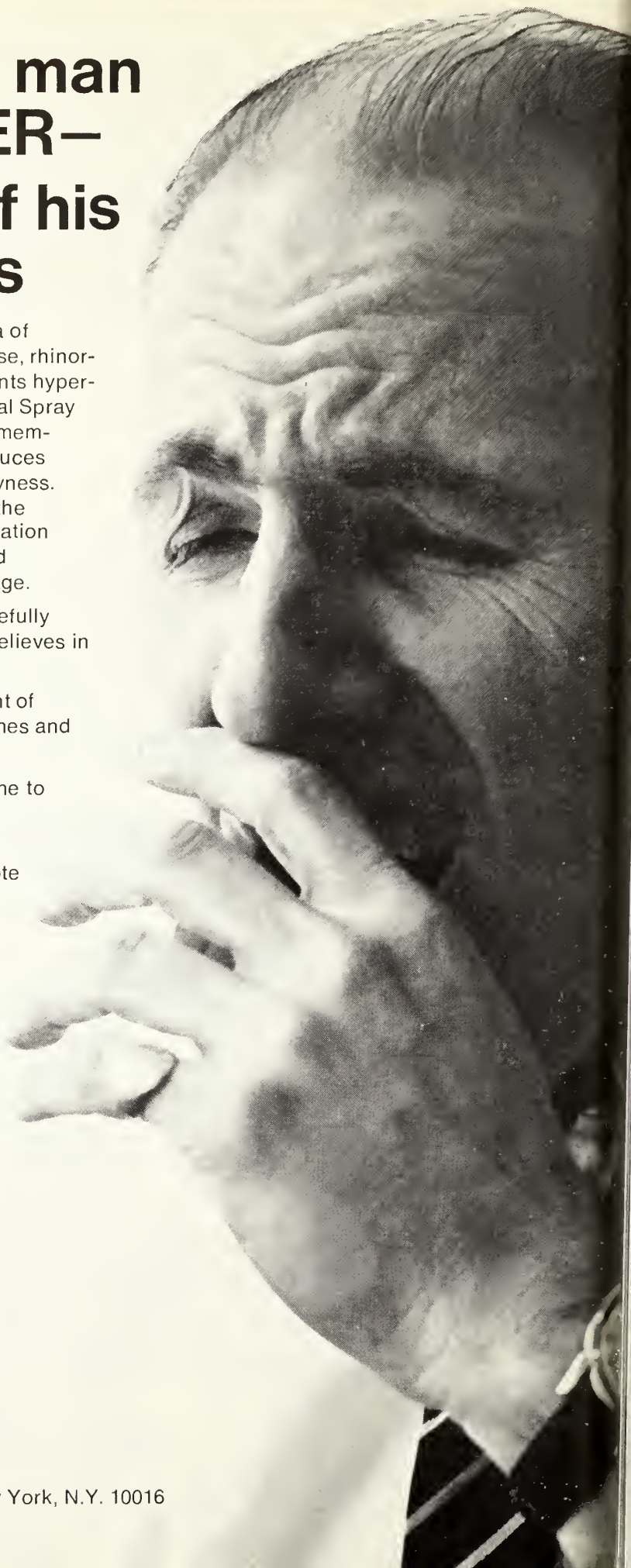
Many times after completion of the combined chemotherapy, patients with extensive scarring, with other associated illnesses, and with chronic bronchial disease may require continued therapy with INH alone.

Single drug treatment, usually carried out with INH is indicated in all cases for prevention or prophylaxis. For indications, see section "Who Should Receive Drugs?" — item #2 — A, B, C, and D and item #3 entitled "potential tuberculosis".

Lifelong follow-up of tuberculosis applies to all patients with known tuberculosis — pulmonary of extra-pulmonary. Such follow-up consists of serial X-rays and periodic bacteriological examinations at first monthly, then every 2 or 3 months. Such follow-up procedures should be reasonably related to the condition of the patient on completion of active therapy. Such measures should also be applied at any time the patient has any respiratory tract or other acute infection, or other departure from his usual state of health.

If a patient with pulmonary tuberculosis denies cough or expectoration either gastric lavages or aerosol cough stimulations should be carried out.

Once merely a man with HAY FEVER— now a victim of his own antibodies



Whatever term describes him in this new era of immunology, the symptoms of congested nose, rhinorrhea and sneezing haven't changed in patients hypersensitive to pollens and molds. But NTZ® Nasal Spray relieves the symptoms. It decongests nasal membranes on contact, relieves itching and reduces excessive rhinorrhea without unpleasant dryness. The first spray of well-tolerated NTZ shrinks the turbinates, helps restore normal nasal ventilation and breathing. After a few minutes, a second spray enhances sinus ventilation and drainage.

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- **T**henfadil® HCl 0.1%, a topical antihistamine to help relieve itching and rhinorrhea.
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Supplied in convenient pocket-size plastic spray bottle of 20 ml. Also available as a solution of 30 ml. (1 fl. oz.) with dropper, and 473 ml. (1 pint).

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AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

New Technique for Cancer of Colon and Rectum

A new technique by which surgeons hope to increase survival rates for cancer of the colon and rectum has been devised and tested over a five-year period by surgeons at the St. Vincent's Hospital and Medical Center of New York.

Survival rates following conventional surgical treatment of this type of cancer have been stubbornly constant over the past twenty years.

While the number of patients treated was too small and the time elapsed too short for statistical significance, survival rates in this study group averaged 15% higher than for patients treated by conventional means. A larger experimental population is needed to determine whether the benefits are real. The procedure involved no unusual risks, the tests showed.

This was reported recently by the American Cancer Society which helped support personnel for the research by Drs. Louis M. Rousselot, Donald R. Cole, John Slattery, Carlo E. Grossi and Eduardo M. Gonzalez.

Combining proven surgical methods, the surgical team sought to destroy stray cancer cells with anti-cancer chemicals just before removing the cancer and the section of the bowel containing it.

Other scientists earlier had found that in a large percentage of cases, cancer cells are dislodged during surgery and they spill into the belly cavity or escape via veins or lymphatic vessels to seed the system with cancer colonies. One study showed cancer cells remained in the abdomen and the operative wound of one-third of the presumed curable cancers; and another showed cancer recurrences in or near the suture lines of 10% of operated patients.

To overcome these hazards, the surgeons at St. Vincent's tied off the digestive tract well above and below the cancer at the beginning of the operation. This prevented cancer cells from escaping from the site. They then injected a powerful anti-cancer drug (Nitrogen Mustard or 5-FU) through the tract wall into the bore of the bowel or rectum. During the next 30 minutes the drug was sopped up by cancer cells in the tumor, in the digestive tract, or in the veins and lymphatics leading from the tract.

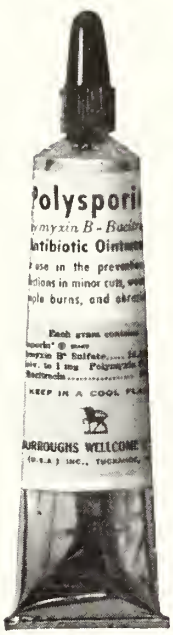
By the time the diseased section of the colon or rectum was removed – a procedure which necessarily involves some handling and the risk of spilling cancer cells – the cancer cells were, hopefully, dead or dying. In any event, cancer recurrence rates after one, two, three and four years were distinctly lower in the experimental group of patients than in other groups.

The number of patients studied in these experiments was 83; they were operated upon between 1960 and 1964. Their destiny was compared with that of a somewhat similar group of 268 patients who underwent surgery – but without drug treatment – between 1956 and 1960.

The American Cancer Society estimates that about 73,000 Americans developed cancer of the colon or rectum in 1965, and about 43,000 died of these cancers. The cause of the cancers is unknown. Slight, if any, progress has been made in curing them. When the cancers are discovered while still seemingly localized (usually they are found on proctoscopic examination), about 60% live five years; when they have spread beyond the original tumor site, fewer than 35% survive five years.

American Cancer Society News Service Release

in treating topical infections, no need to sensitize the patient



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Maine Heart Association Notes

116 State Street, Augusta, Maine

DeBakey Will Keynote Maine Heart Association's Annual Meeting To Be Held In September

Michael DeBakey, M.D., nationally known cardiovascular surgeon of Houston, Texas, will be the keynote speaker at the Annual Meeting of the Maine Heart Association to be held at the Sheraton Eastland Motor Hotel, Portland, Saturday, September 10, according to Harold L. Osher, M.D., vice-president of the Association and chairman for the annual event.

Dr. DeBakey will speak at the luncheon to be held after the Scientific Sessions scheduled for the morning.

Dr. Osher suggested that due to the great interest in Dr. DeBakey's work, those wishing to hear the surgeon should make early reservations. Registration cards can be obtained by writing the Executive Director, Maine Heart Association, 116 State Street, Augusta, Maine.

113TH ANNUAL SESSION OF THE M.M.A. HOUSE OF DELEGATES — *Continued from Page 172*

District (Cumberland and York Counties) and Charles F. Branch, M.D. of Lewiston, Councilor for the Second District (Androscoggin, Franklin and Oxford Counties). Both of these appointments are for a three-year term expiring in 1969.

Annual Reports — Reports were submitted prior to the meeting by the following and included in the House of Delegates folder:

Paul S. Hill, Jr., M.D., Councilor and Council Chairman; and Councilors, John D. Denison, M.D., James C. Bates, M.D. and Asa C. Adams, M.D.

Standing Committees: Legislative, Kevin Hill, M.D. and Public Relations, Charles R. Glassmire, M.D.

Special Committees: Diabetes, Melvin Bacon, M.D.; Maternal and Child Welfare, Alice A. S. Whittier, M.D.; Advisory Committee to Implement the Kerr-Mills Bill in the State of Maine, Philip P. Thompson, Jr., M.D.; Maternal Mortality, Robert M. Knowles, M.D.; Maine Committee – AMA-ERF, Charles R. Glassmire, M.D.; Problems of Long-Term Patient Care, Peter W. Bowman, M.D.; Medicine and Religion, Asa C. Adams, M.D.; Disaster Medical Care, John T. Konecki, M.D.; Blood Transfusion, Joseph E. Porter, M.D.; Amy W. Pinkham Fund, Virginia C. Hamilton, M.D.; Arthritis, Philip P. Thompson, Jr., M.D.; and Liaison Activities Between the Maine State Nurses' Association and the M.M.A., George O. Chase, M.D.

The report of the delegate to the Connecticut State Medical Society, Robert M. Knowles, M.D. and the report by the Secretary-Treasurer, Esther M. Kennard, were also included in the Delegate's folder.

Reports were presented at the meeting by the Executive Director, Daniel F. Hanley, M.D.; Charles F. Branch, M.D., Councilor; Thomas A. Martin, M.D., Chairman, Medical Advisory Committee; Francis A. Winchenbach, M.D., Chairman, Health Insurance Committee; Dexter J. Clough, 2nd, M.D., Chairman, Committee on Conservation of Vision; Charles A. Hannigan, M.D., Chairman, Medical School for Maine Committee; and Charles D. McEvoy, Jr., M.D., Chairman, Advisory Committee to the Health Facilities Planning Council of Maine. Reports were also presented by the following delegates to out-of-state medical societies: Harland G. Turner, M.D., Massachusetts; Paul S. Hill, Jr., M.D., Vermont-New Hampshire combined meeting; John F. Dougherty, M.D., New York; and Leonard G. Miragliuolo, M.D., Rhode Island. John B. Truslow, M.D. presented a report concerning the Medical School for Maine Feasibility Study.

Presentation of A. H. Robins Award — The Community Service Award for 1966, sponsored by the A. H. Robins Company, was presented to Francis J. O'Connor, M.D. of Augusta by John F. Dougherty, M.D., M.M.A. President.

Presentation of Historical Paintings — Mr. W. E McCormick, New England Manager of Parke, Davis and Company, presented a set of four framed pictures of a "History of Medicine" series to Dr. Dougherty, M.M.A. President; these paintings are now in the office of the State Association.

Special Memberships — The recommendations for special memberships were approved. (The list of members recommended for special memberships was included in the Delegate's folder.)

Guests — The guest list included Mrs. Benjamin L. Shapiro, President, Woman's Auxiliary to M.M.A.; B. Albert Ring, M.D., delegate from Vermont Medical Society; Donald A. Nickerson, M.D., delegate from Massachusetts Medical Society; Thurman B. Givan, M.D., delegate from New York Medical Society; John B. Truslow, M.D., Medical School for Maine Feasibility Study; William T. Strauss, M.D., Albany Medical School; George J. Robertson, M.D., Bingham Associates; Mr. Maurice A. Kramer, AMA Field Representative; Mr. Richard F. Nellson, Executive Director, Associated Hospital Service of Maine; Mr. Robert Cash, Union Mutual Life Insurance Company; and Mr. W. E. McCormick, New England Regional Manager, Parke, Davis and Company.

Stenographic Record — A summary of the proceedings of the House of Delegates is being sent to the county presidents and secretaries and to the delegates and alternates of each county society. (The complete report is on file in the Association's office in Brunswick, where it is available to any member of the Association.)

General Assembly

The General Assembly of the Maine Medical Association was called to order by the President, John F. Dougherty, M.D. at 4:00 P.M. on Monday, June 13, 1966.

Election of President-elect — At this session, Paul S. Hill, Jr., M.D. of Saco, was elected President-elect.

Presentation of the Mead Johnson Laboratories' Aesculapius Award for the Outstanding Scientific Exhibit was made to Richard C. Dillihunt, M.D. of Portland.

President's Address — Dr. Dougherty delivered his President's Address at this General Assembly.

ESTHER M. KENNARD
Secretary-Treasurer, M.M.A.

News, Notes and Announcements

Mrs. Anton Elected AMA Auxiliary Director



Mrs. Thomas Anton of Old Orchard Beach was elected to a two-year term on the Board of Directors of the Woman's Auxiliary to the American Medical Association during the Auxiliary's 43rd Annual Convention in Chicago.

A past president of both the Maine and York County auxiliaries, Mrs. Anton is also a past president of the Webber Hospital Auxiliary in Biddeford and currently chairman of the hospital's Candy-Striper Program. She has just completed a term as Eastern regional program chairman of the AMA Auxiliary.

Dr. Loring Appointed Chief Pathologist at Mercy Hospital

William E. Loring, M.D., who has been in charge of the Columbia University Division of Pathology at Bellevue Hospital, New York City, and visiting pathologist, St. Vincent's Hospital, Bridgeport, Connecticut, has been appointed chief pathologist at Mercy Hospital.

Dr. Loring has had more than 20 technical articles published in medical and scientific journals and periodicals. He is a member of several state and national professional organizations and holds many postgraduate awards.

In his new position, Dr. Loring will direct Mercy Hospital's laboratory and participate in the hospital's educational programs.

Dr. Darlington Named Director to National Tuberculosis Association

Brinton T. Darlington, M.D. of Augusta was elected a director-at-large of the National Tuberculosis Association at its annual meeting in San Francisco, California.

Dr. Darlington is a former president of the Maine Thoracic Society and the Maine Tuberculosis and Health Association and is president-elect of the Maine Society of Internal Medicine.

Dr. Perkins Named Director of New State Bureau

Niles L. Perkins, M.D. of Portland has been named director of a new Bureau within the State Department of Health and Welfare, the Bureau of Medical Care, in a recent announcement made by Dr. Dean Fisher.

"This Bureau," said Dr. Fisher, "will have the immediate responsibility of developing further our welfare medical care programs, developing and applying the necessary administrative procedures, program information and interpretation, staff training, program integration and extension."

Institute on "Team Work for the Chronically Ill"

An Institute on "Team Work for the Chronically Ill", sponsored by the Maine Department of Health and Welfare, will be held at the Shawmut Inn in Kennebunkport, Maine on September 21-23, 1966.

The three-day Institute will include the following topics and speakers:

September 21

"Greetings and Opening Remarks" – Dean Fisher, M.D.; "Chronic Disease Care – 1966, and the Professions" – William L. Kissick, M.D.; "Team Work, What, How and Where" – Mrs. Evelyn Cooper; "Team Work in Action – The Hospital Administered Home Care Team" – Moderator, Miss Eileen Lester; and "Communication and the Team" – Sol Levine, Ph.D.

September 22

"Team Work in Action – The Community Based Home Care Program" – Moderator, Miss Eileen Lester; "Team Work in Action – An Extension of Hospital Care Through Nursing Homes and Home Care Services" – Moderator, Mrs. Ruth Taylor; "Team Work in Rural Maine" – Moderator, Miss Mary Sullivan; and "Medicine and Team Work" – Ellsworth T. Newmann, M.D.

September 23

"Team Work – Rehabilitation Services in a Maine Hospital" – Harold Willard, M.D., Price Kirkpatrick, M.D. and Mrs. Mary Dionne; "Team Work – The Professions and the Community" – Claire F. Ryder, M.D.; and "Findings and Recommendations of Group Discussions" – Miss Agnes Flaherty.

Third National Congress on Medical Quackery

The Third National Congress on Medical Quackery will be held October 7-8, 1966 at the Pick-Congress Hotel in Chicago.

Joint announcement of the Congress was made by F. J. L. Blasingame, M.D., executive vice-president of the American Medical Association, and Peter G. Meek, executive director of The National Health Council.

The AMA and The National Health Council, the nation's largest organization of professional, governmental and voluntary agencies in the health field, will serve as co-sponsors of the October Congress.

The October Congress will be based on the theme of "Quackery: 1966" and will be aimed at calling to the attention of the nation the perils posed by present-day fads and fallacies in the health field.

Details of the Congress, including the program of nationally-recognized speakers, will be announced in the near future. In-

quiries should be addressed to John G. Thomsen, M.D., Chairman, American Medical Association, Committee on Quackery, 535 N. Dearborn St., Chicago, Illinois 60610.

Reserve the re-scheduled dates now New England Postgraduate Assembly January 17-19, 1967 Statler Hilton Hotel, Boston, Massachusetts

Advance Program will be sent to all New England physicians in early December.

Lakeside Laboratories Offers Film and Self-Rating Scale on Measurement of Depression

"THE MEASUREMENT OF DEPRESSION" is a film depicting the development, validation, and use of a scale for the quantitative measurement of depression.

The scale was designed by Dr. William W. K. Zung, Durham, N.C., psychiatrist. Although initially devised for use in psychiatric research, it readily lends itself to the general practice of medicine where most depressions are first encountered.

The film is designed for medical educational purposes at meetings of hospital staffs, county societies or specialty groups. The self-rating scales, available in quantity for use in office practice, come complete with full instructions.

Both the film and pads of the self-rating scale are available free upon request from Lakeside Laboratories, Inc., Milwaukee, Wisconsin 53201.

The scale consists of a list of 20 statements expressed in the common language of the patient. The statements comprehensively delineate widely recognized symptoms of depression including disturbances of mood, biological and psychological function. Testing and scoring patients usually requires less than five minutes.

FILM INFORMATION

Running Time: 22 minutes, 16 mm., sound and color
Produced by: Sturgis-Grant Productions, Inc.
Sponsored by: Lakeside Laboratories, Inc.
The film is available on loan without charge.
To schedule a showing of "THE MEASUREMENT OF DEPRESSION", please see your Lakeside Professional Service Representative or write or phone:

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1707 East North Avenue
Milwaukee, Wisconsin 53201

New Members of the Maine Medical Association

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Charles H. Johnson, M.D., 73 Fraser St., Edmundston, N.B., Canada
Frank J. Twadelle, M.D., 5 High St., Van Buren

CUMBERLAND

Edmund W. Hardy, M.D., 134 U.S. Rt. 1, Falmouth
John E. Knowles, M.D., 131 Chadwick St., Portland
Houghton M. White, M.D., 56 Baribeau Dr., Brunswick

KENNEBEC

Efraim C. Galarraga, M.D., Box 724, State Hospital, Augusta
Chandra Lal, M.D., Box 724, State Hospital, Augusta
Dimitri Polydefkis, M.D., Box 724, State Hospital, Augusta
Ahmet Satir, M.D., Box 724, State Hospital, Augusta
Paul Stucki, M.D., 72 Elm St., Waterville

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Contraindications: In hyperexcitability and in agitated prepsychotic states. Previous allergic or idiosyncratic reactions.

Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised; especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of pre-existing symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dose—operation of motor vehicles, machinery or other activity requiring alertness should be avoided. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia, the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor, and respiratory collapse.



First aid for a button popper



Second aid for a button popper

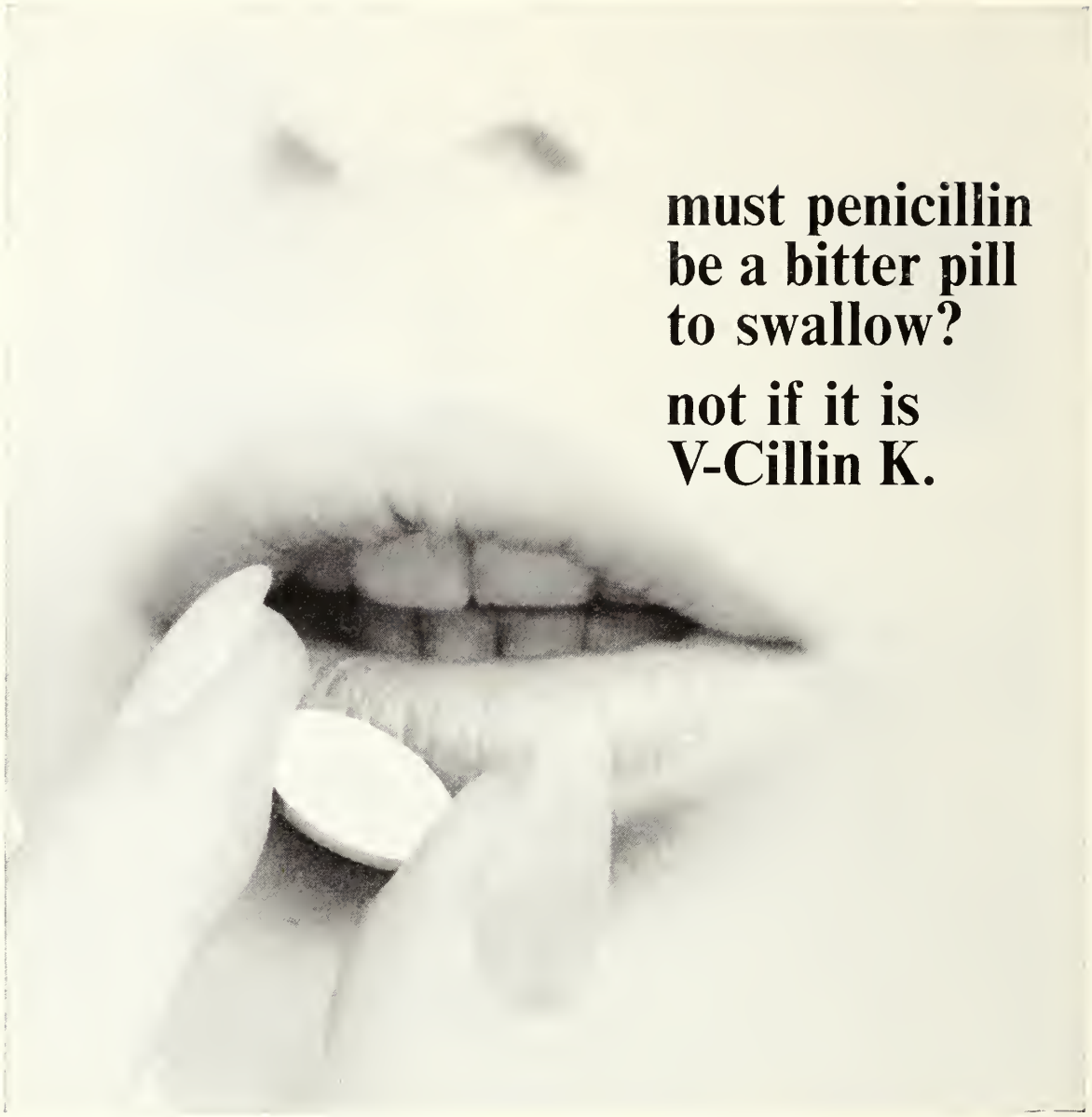
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Indications: V-Cillin K is an antibiotic useful in the treatment of infections caused by streptococci, pneumococci, and sensitive strains of staphylococci.

Contraindications and Precautions: Although sensitivity reactions are much less common after oral than after parenteral administration, V-Cillin K should not be administered to patients with a history of allergy to penicillin. As with any antibiotic, observation for overgrowth of nonsusceptible organisms during treatment is important.

Usual Dosage Range: 125 mg. (200,000 units) three times a day to 250 mg. every four hours.

Supplied: Tablets V-Cillin K, 125 or 250 mg.; also, V-Cillin K, Pediatric, 125 mg. per 5-cc. teaspoonful, in 40, 80, and 150-cc.-size packages.

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The human spine is not engineered for prolonged sitting at desks, pianos, typewriters and drafting boards. The stresses set up by the heavy, forward-tilted head and trunk, balanced precariously on an insufficient base, result in strain of the dorsal musculature, particularly at the low lumbar level.

The unusual muscle-relaxant and analgesic properties of 'Soma' make it especially useful in the treatment of low back sprains and strains. 'Soma' is widely prescribed ☐ **to relieve pain** ☐ **to relax muscles** ☐ **to restore mobility.**

Indications: 'Soma' is useful for management of muscle spasm, pain, and stiffness in a variety of inflammatory, traumatic, and degenerative musculoskeletal conditions. It also may act to normalize motor activity in certain neurologic disturbances.

Contraindications: Allergic or idiosyncratic reactions to carisoprodol.

Precautions: 'Soma', like other central nervous system depressants, should be used with caution in patients with known propensity for taking excessive quantities of drugs and in patients with known sensitivity to compounds of similar chemical structure, e.g., meprobamate.

Side Effects: The only side effect reported with any frequency is sleepiness, usually on higher than recommended doses. An occasional patient may not tolerate carisoprodol because of an individual reaction, such as a sensation of weakness. Other rarely observed reactions have included dizziness, ataxia, tremor, agitation, irritability, headache, increase in eosinophil count, flushing of face, and gastrointestinal symptoms.

One instance each of pancytopenia and leukopenia, occurring when carisoprodol was administered with other drugs, has been reported, as has an instance of fixed drug eruption with carisoprodol and subsequent cross reaction to meprobamate. Rare allergic reactions, usually mild, have included one case each of anaphylactoid reaction with mild shock and angioneurotic edema with respiratory difficulty, both reversed with appropriate therapy. In cases of allergic or hypersensitivity reactions, carisoprodol should be discontinued and appropriate therapy initiated. Suicidal attempts may produce coma and/or mild shock and respiratory depression.

Dosage: Usual adult dose is one 350 mg. tablet three times daily and at bedtime.

Supplied: Two Strengths: 350 mg. white tablets and 250 mg. orange, two-piece capsules.

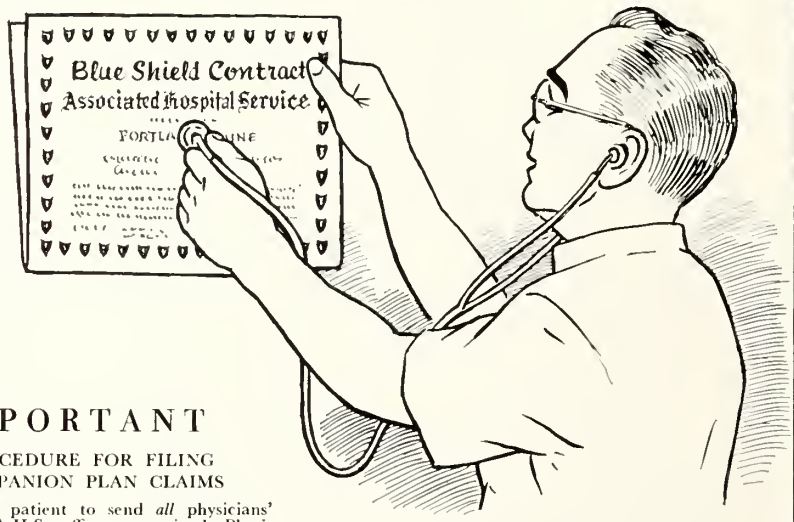
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Instruct your patient to send *all* physicians' bills to the A.H.S. office as received. Physician services must be itemized and their "reasonable charges" for each item indicated on the bills. A brief description of the injury or illness treated should also be shown. *The Companion Plan contract number and the Social Security or Government Identification number must be entered on the bill by the patient or the doctor.* For prompt direct payments of the Companion Plan benefits the bill should be issued to the patient in duplicate, one for Blue Cross-Blue Shield use and one for the Part B intermediary if you are not making claim on the Government Form SSA 1490.

If the bill is not receipted, payment will be made directly to the doctor on the regular semi-monthly Blue Shield check. The notation "C.P." for Companion Plan will be shown under the heading "Coverage" on the check voucher. This procedure will assure the doctor of the most prompt payment.

If the bill is receipted, payment of benefits will be made to the patient.

Medicare "Explanation of Benefits Form" may be used by the patient making claim for benefits but should be accompanied by itemized physicians' bills for all services incurred during the calendar year.

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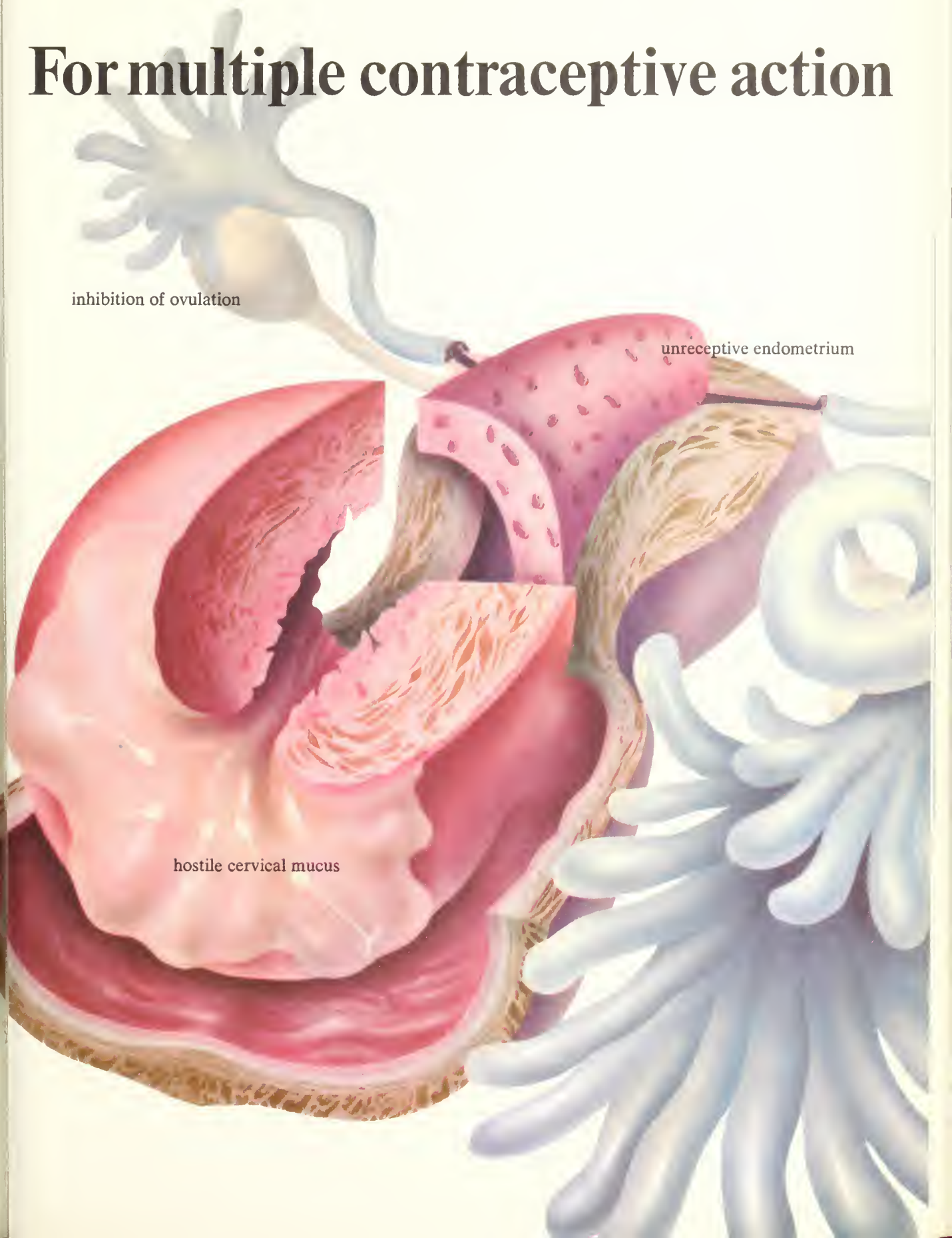
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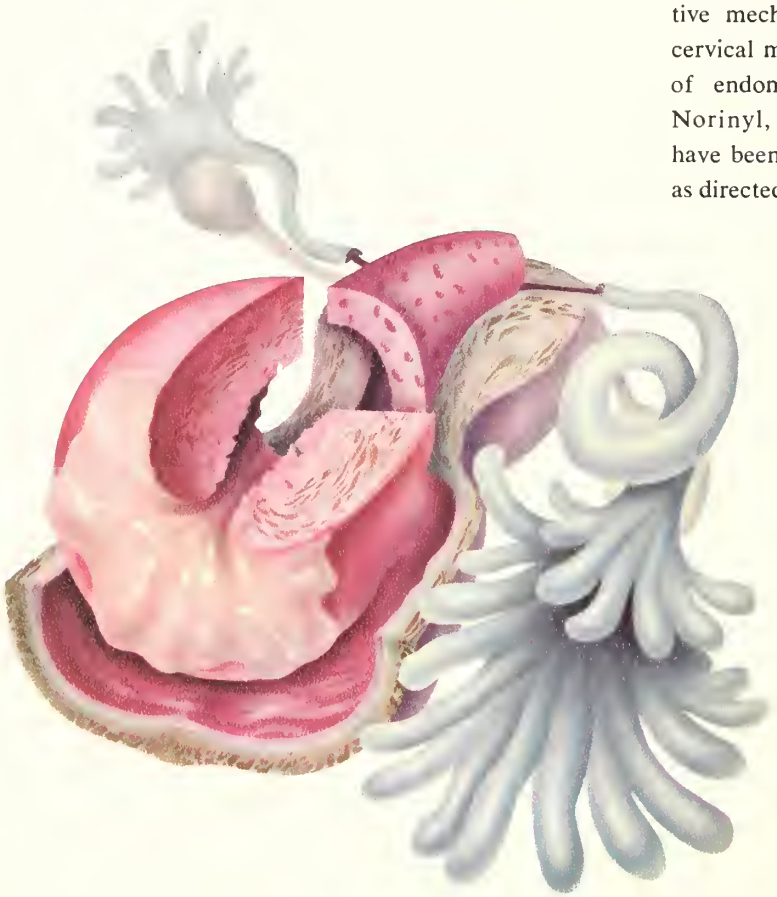
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multiple action that has produced a record of unexcelled effectiveness

inhibition of ovulation by means of 2 time-proved hormonal agents

production of a cervical mucus hostile to sperm motility and vitality

creation of an endometrium unreceptive to egg implantation



no unplanned pregnancies

Norinyl provides multiple action for maximum assurance of success. It does not depend on ovulation inhibition alone for contraceptive effectiveness. The mechanism of action of combined hormonal therapy results in ovulation inhibition reinforced by other protective mechanisms, including a hostile cervical mucus[1-13] and an acceleration of endometrial changes.[1-3,7-16] With Norinyl, no unplanned pregnancies have been reported to date when used as directed.

plus important supportive benefits that help her through those critical early months of oral contraception

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Low incidence of BTB and spotting, nausea and amenorrhea tends to minimize side effect problems and increases patient cooperation.

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An unbreakable "confusionproof" package makes it easy to adhere to prescribed dosage schedule: individually sealed tablets numbered from 1 through 20 *plus* monthly calendar record enables patient to double-check dosage intake by day and corresponding tablet number.



Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

References: 1. Council on Drugs: JAMA 187:664 (Feb 29) 1964. 2. Bryans, F. E.: Canad. Med. Ass. J. 92:287 (Feb. 6) 1965. 3. Goldzieher, J. W.: Med. Clin. N. Amer. 48:529 (Mar.) 1964. 4. Cohen, M. R.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med. Sci. 16:26 (Nov.) 1965. 5. Hammond, D. O.: Ibid. 6. Rice-Wray, E., Goldzieher, J. W., and Aranda-Rosell, A.: Fertil. Steril. 14:402 (Jul.-Aug.) 1963. 7. Goldzieher, J. W., Moses, L. E., and Ellis, L. T.: JAMA 180:359 (May 5) 1962. 8. Kempers, R. D.: GP 29:88 (Jan.) 1964. 9. Tyler, E. T.: JAMA 187:562 (Feb. 22) 1964. 10. Rudel, H. W., Martinez-Manautou, J., and Maqueo-Topete, M.: Fertil. Steril. 16:158 (Mar.-Apr.) 1965. 11. Flowers, C. E., Jr.: N. Carolina Med. J. 25:139 (Apr.) 1964. 12. Goldzieher, J. W.: Appl. Ther. 6:503 (June) 1964. 13. The Control of Fertility. Report adopted by the Committee on Human Reproduction of the American Medical Association. JAMA 194:462 (Oct. 25) 1965. 14. Flowers, C. E., Jr.: JAMA 188:1115 (June 29) 1964. 15. Merritt, R. I.: Appl. Ther. 6:427 (May) 1964. 16. Newland, D. O.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med. Sci. 16:26 (Nov.) 1965.

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*As many as 9 out of 10 urinary tract infections are now caused by gram-negative organisms: E. coli, Klebsiella, Aerobacter, Proteus, Paracolon or Pseudomonas[2]... However, infections of the urethra and prostate caused by non-gonococcal gram-negative organisms are believed to be less prevalent.

tions: Urinary tract infections caused by gram-negative and some gram-e organisms.

effects: Mainly mild, transient gastrointestinal disturbances; in onal instances, drowsiness, fatigue, pruritus, rash, urticaria, mild ophilia, reversible subjective visual disturbances (overbrightness of change in visual color perception, difficulty in focusing, decrease in acuity and double vision), and reversible photosensitivity reactions. d overdosage, coupled with certain predisposing factors, has produced onvulsions in a few patients.

utions: As with all new drugs, blood and liver function tests are advis-uring prolonged treatment. **Pending further experience, like most therapeutic agents, this drug should not be given in the first trimester gnancy. It must be used cautiously in patients with liver disease or impairment of kidney function.** Because photosensitivity reactions have ed in a small number of cases, patients should be cautioned to avoid essary exposure to direct sunlight while receiving NegGram, and if a on occurs, therapy should be discontinued. The dosage recommended adults and children should not arbitrarily be doubled unless under the l supervision of a physician. Bacterial resistance may develop.

testing the urine for glucose in patients receiving NegGram, Clinistix® nt Strips or Tes-Tape® should be used since other reagents give a positive reaction.

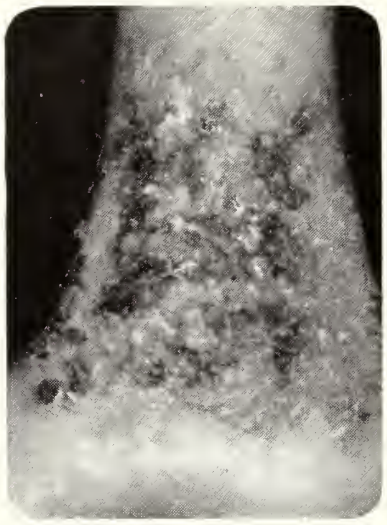
e: Adults: Four Gm. daily by mouth (2 Caplets® of 500 mg. four times for one to two weeks. Thereafter, if prolonged treatment is indicated, sage may be reduced to two Gm. daily. Children may be given ximately 25 mg. per pound of body weight per day, administered in d doses. The dosage recommended above for adults and children l not arbitrarily be doubled unless under the careful supervision of a ian. Until further experience is gained, infants under 1 month l not be treated with the drug.

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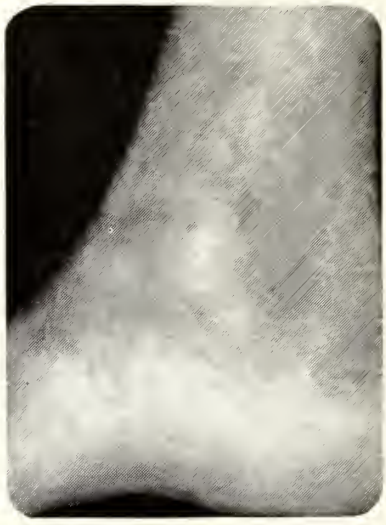
ences: (1) Based on 23 clinical papers, 1512 cases. Bibliography on st. (2) Bush, I. M., Orkin, L. A., and Winter, J. W., in Sylvester, J. C.: icrobial Agents and Chemotherapy—1964, Ann Arbor, American ty for Microbiology, 1965, p. 722.

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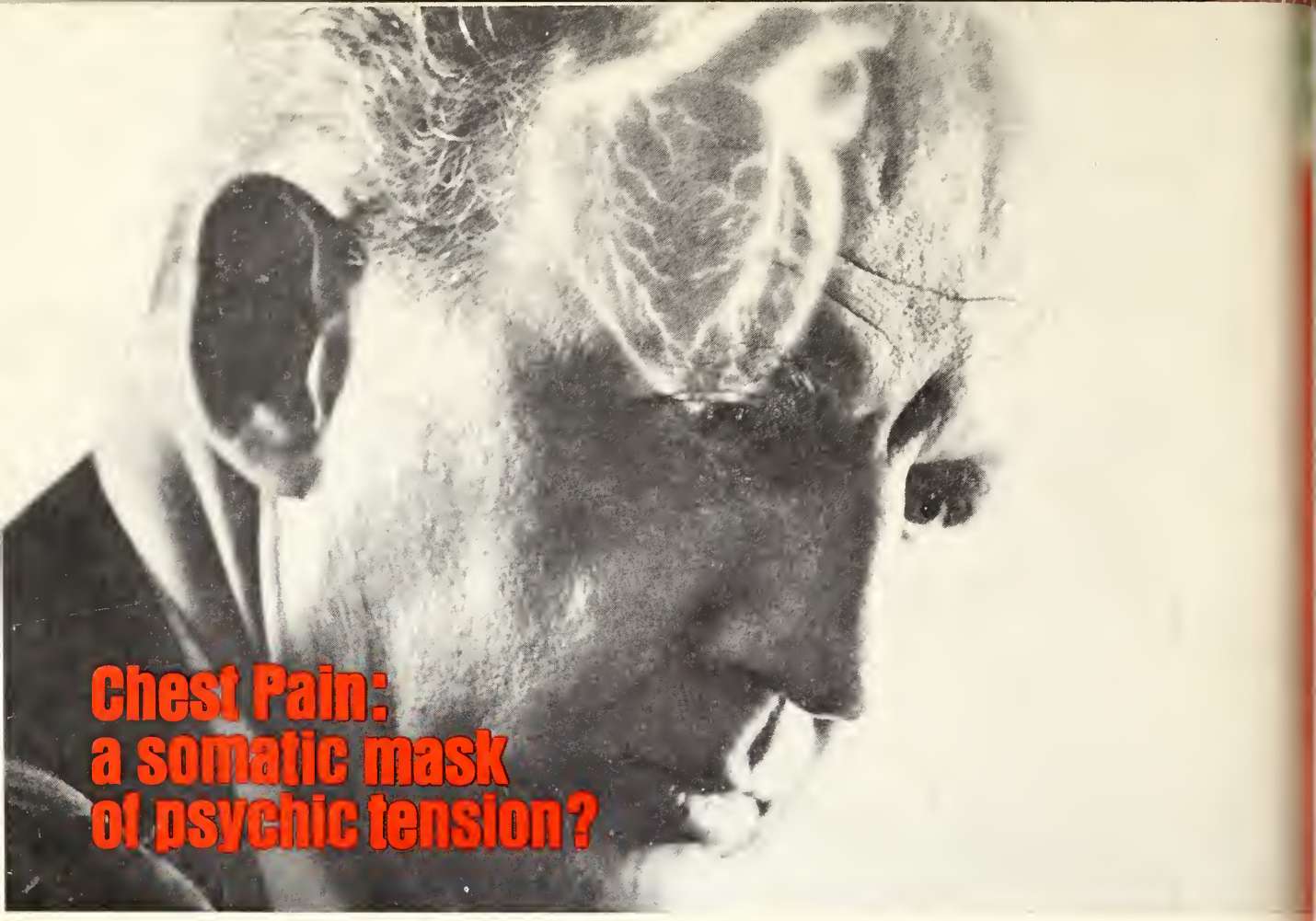
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Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

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THE JOURNAL
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VOLUME 57 SEPTEMBER, 1966 NUMBER 9

Maine Medical Center Number

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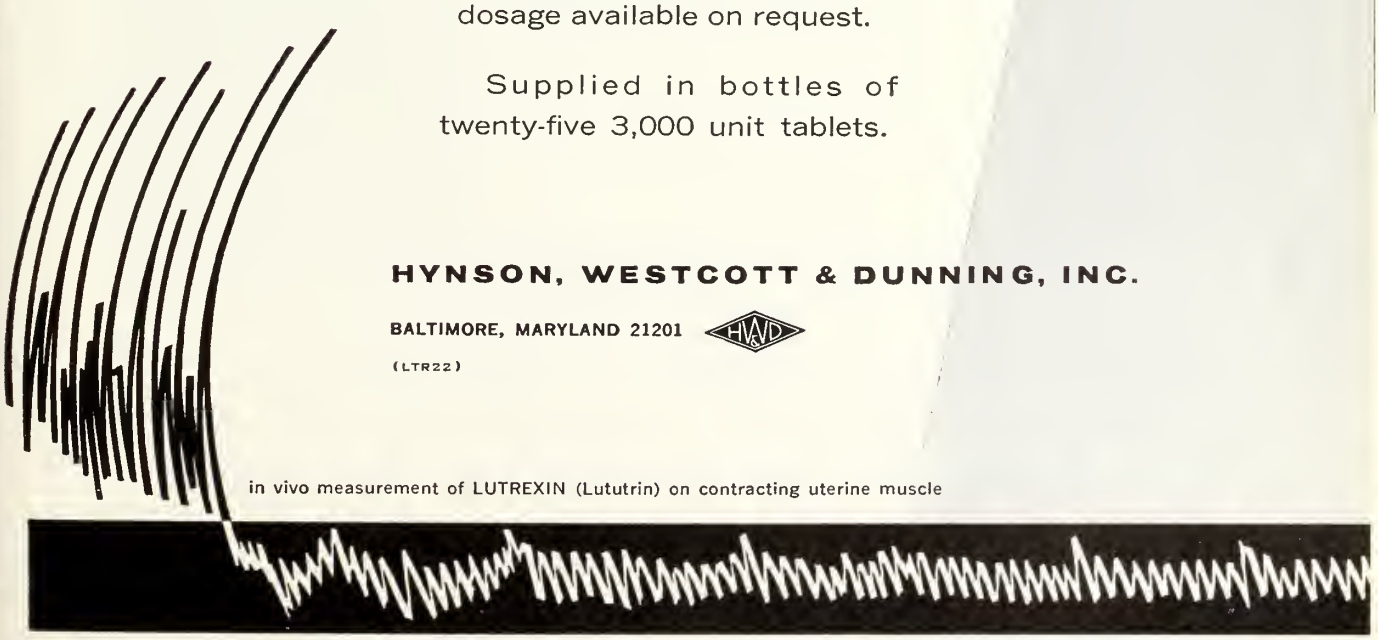
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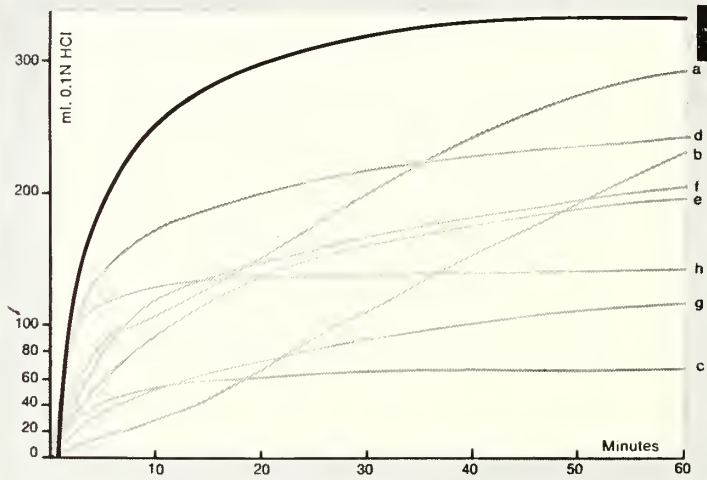
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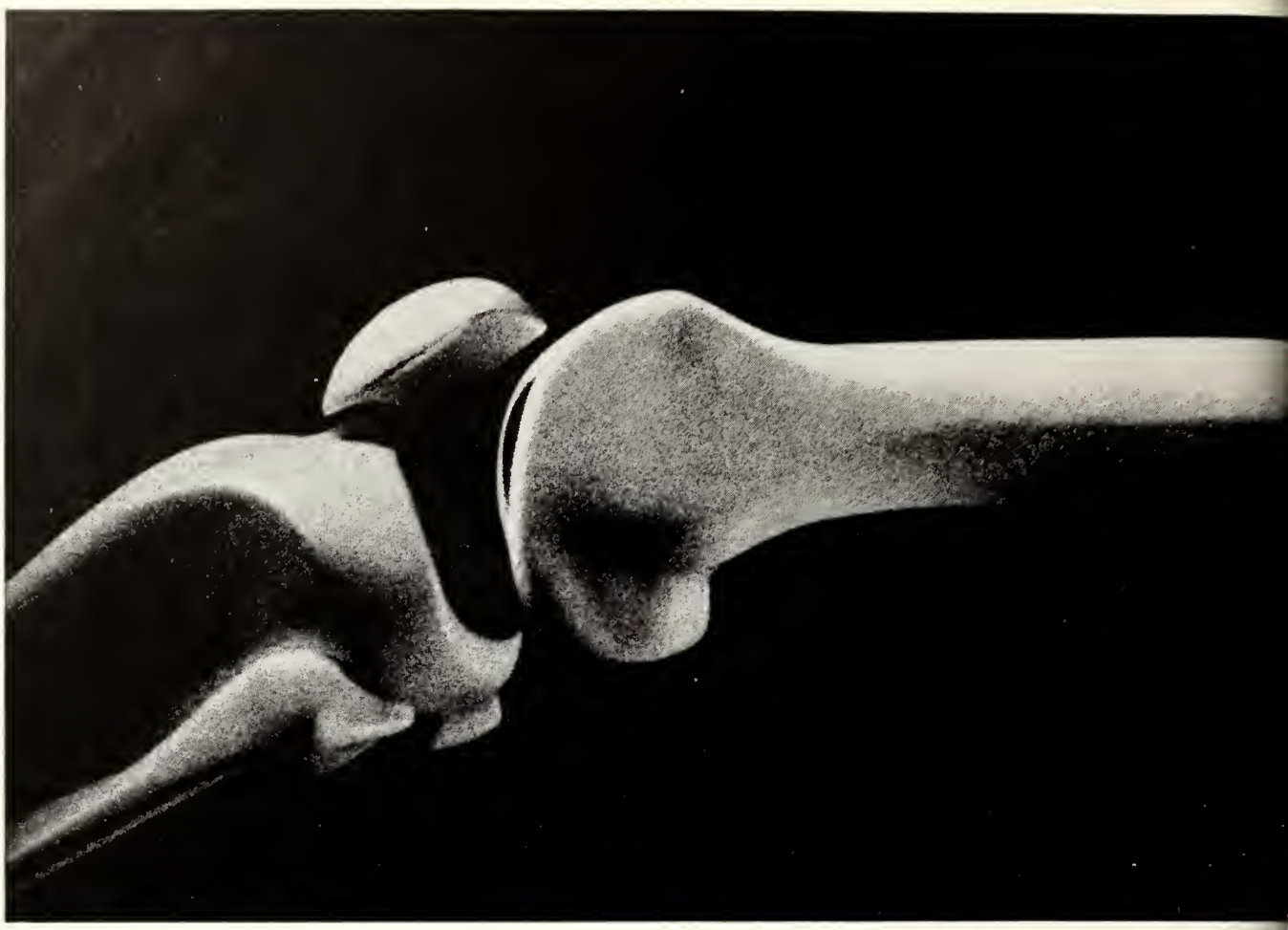
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Before prescribing, the physician should obtain a detailed history and perform a complete physical and laboratory examination, including a blood count. The patient should be kept under close supervision and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Regular blood counts should be made to guard against blood dyscrasias.

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The most common adverse reactions are nausea, edema and drug rash. Moderately lowered red cell count may sometimes occur due to hemodilution. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome or a generalized allergic reaction may occur and require withdrawal of medication. Stomatitis, salivary gland enlargement, vertigo or languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia are also possible side effects.

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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

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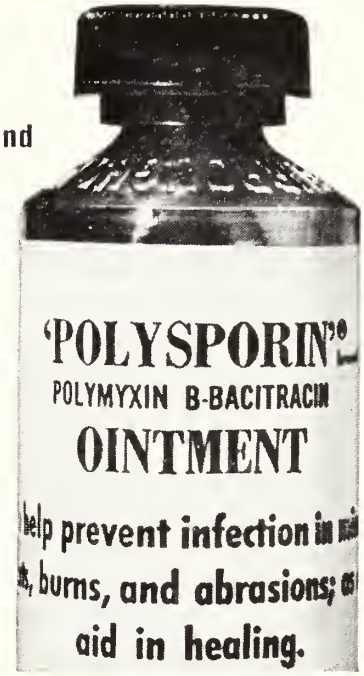
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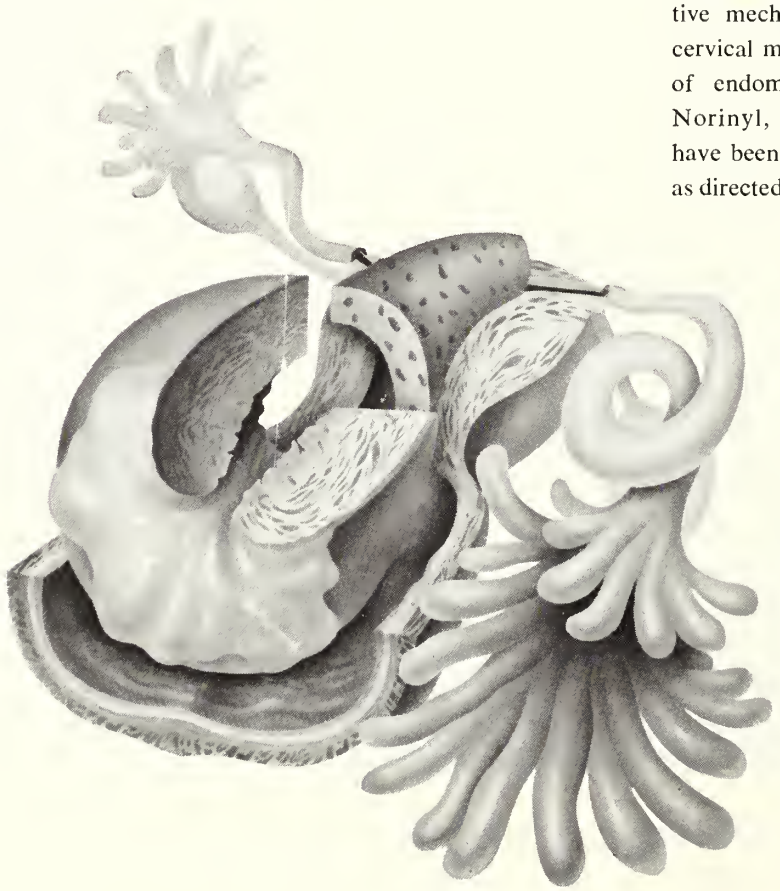
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Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

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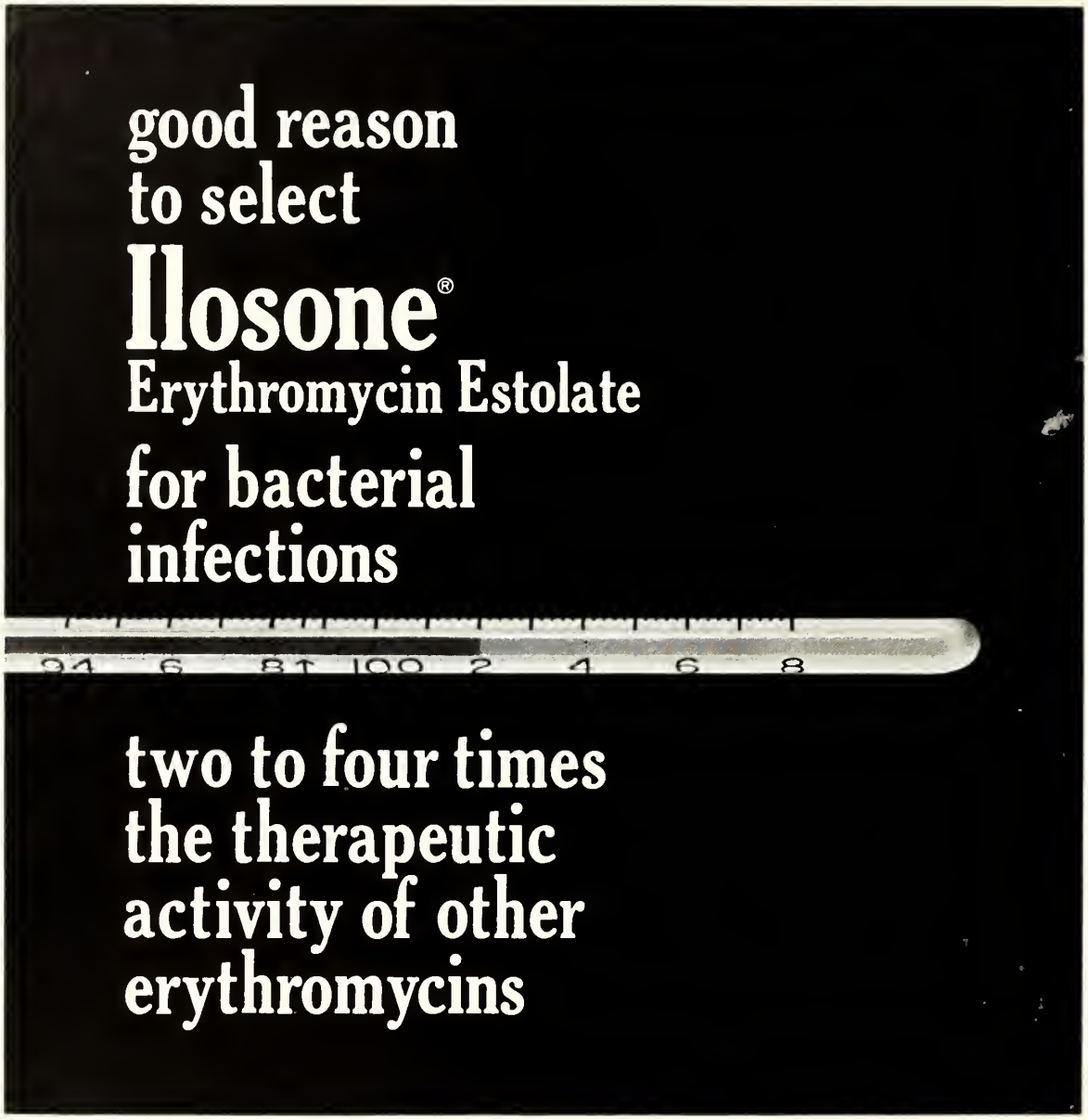
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Hemophilia: Old and New*

Louis G. Bove, M.D.

"The first person of whom I have any account, as being subject to the remarkable predisposition to be described, was Mr. Oliver Appleton of Ipswich, about one hundred years since. This man was subject from his youth to profuse bleeding from slight causes. When advanced in life, by a long confinement in bed the skin was worn from his hips and an hemorrhage taking place from that part and from the urethra, occasioned his death. He left three daughters, two of whom married into a family by the name of Swain, in Reading. Dr. Thomas Swain who married the eldest had by her two sons and five daughters. The two sons both bled to death. During their lives they were liable to violent hemorrhages from the smallest scratch or injury, the bleeding usually coming on about a week after they were hurt."[1] This paragraph, taken from an article entitled, *"Account of a Remarkable Hemorrhagic Disposition"* by Dr. John Hay of Reading, Massachusetts, appeared in the *New England Journal of Medicine and Surgery* in 1813 and is still the classic description of the disease hemophilia. It followed by ten years the original description of the hereditary aspects of the disease, these observations being made by John Conrad Otto, a young Philadelphia physician.

Few diseases have been dramatized as much as hemophilia, the royal disease, the curse of the Hapsburg. This problem, however, is not limited to any country or family. One needs only to look about, and the hemophiliac patient is always there. We are not only interested in the out-and-out classical hemophiliac, but as well in the so-called mild hemophiliac who may live into later life without a serious hemorrhage until he or she is subjected to an elective or emergency surgical procedure. I would like to remind you of some of the old sound and true concepts of this disease as well as to acquaint you with some of the newer developments in diagnosis and treatment.

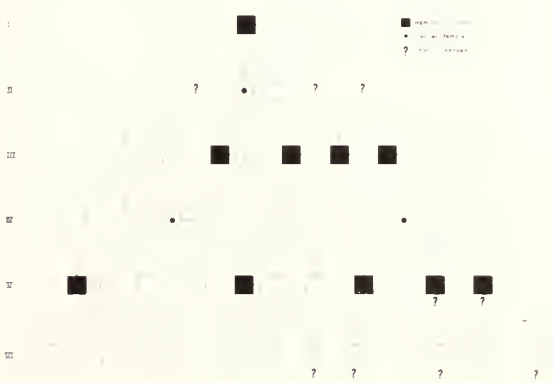


Fig. 1

Hemophilia is an inherited disease characterized by defective coagulation of the blood which results in excessive hemorrhage after injury.

This disease is transmitted as a sex-linked recessive trait. That means that if a hemophiliac marries a normal woman, all his sons will be entirely normal, whereas all his daughters will be carriers, inherit the defect but will not be bleeders. They in turn transmit this defect to half their sons and daughters. The sons will be bleeders, and the daughters again will be carriers (Fig. 1).

A word of caution, however; sporatic hemophilia,[2] this is the appearance of the disease in a patient without a family history, is quite common. Evidence suggesting mutation is convincing. Generally, sex-linked recessive diseases tend to die out by dilution; and the incidence of hemophilia remains fairly constant as 1:20,000 births. Hence, do not disregard a suggestive history on the basis that there is no family history of a bleeding tendency.

As originally stated, this disease is due to defective coagulation of the blood. There are at least twelve and

*Presented at Alumni Day, Maine Medical Center, Portland, Maine on May 12, 1966.

probably more separate constituents of the blood required for this phenomenon.[3] The congenital hemophiliac is now known to be of three distinct types, depending upon which of these factors he or she is deficient in. The classical hemophiliac lacks a protein called anti-hemophilicglobulin (AHG) or Factor VIII. The next most common type, originally referred to as Christmas Disease (named after the first patient in England described with this affliction), is due to a deficiency of plasma-thromboplastin compliment (PTC) or Factor IX. The third hemophiliac syndrome has no proper name and is due to a deficiency of plasma-thromboplastin-antecedent (PTA) or Factor XI.

Both classical hemophilia and Christmas Disease follow the same genetic pattern.[4] PTA deficiency or Factor XI does not. It is transmitted as an autosmal dominent trait, and thus is present in both men and women. A much more rare syndrome, it is present predominently in the Jewish race. Hemarthrosis is uncommon, and the principal danger is in postoperative hemorrhage. With this exception it is not the type of hemophilia which determines the severity of the disease but the severity of the deficiency of the specific factor involved. Normal levels of AHG may vary from 50-110% whereas in the patient affected the level may vary from 1-30.% A level of 1% associated with the severe disease and that of 20-30% the mild syndrome. Carrier females have levels of about 50%, and assaying levels of AHG has provided a method of determining whom a carrier might be.

The importance of making the correct diagnosis not only as to the hemophiliac syndrome, but to the specific factor deficient is a necessity because of a difference in treatment. The AHG or Factor VIII is present only in fresh blood or fresh frozen plasma, and it is very labile with a half life of eight to twelve hours. PTC or Factor IX, however, is quite stable and is present in refrigerated stored plasma with a half life of twenty-four to thirty-six hours. PTA or Factor XI is present in both fresh and stored plasma and has a half life of two to three days. Hence, knowing the specific factor involved is a must if adequate, but not excessive, therapy is to be given.

The problem of making the diagnosis, first of the defect in coagulation and then of the specific type of hemophiliac syndrome, is in a constant flux depending upon the many new and sensitive laboratory tests available. We have come a long way from the simple "bleeding and clotting time" work-up of days past. Now we have a simple group of tests applicable to every laboratory which will pick up the mild hemophiliac and more complex tests done in the larger laboratories which can differentiate the specific type. Also assay methods are available which help tremendously in the management of the hemophiliac patient through a bleeding episode. The importance of these new tests is recognized when one realizes that at levels of AHG above 1% the clotting time will be normal!

Fig. 2 outlines the three stages of coagulation and

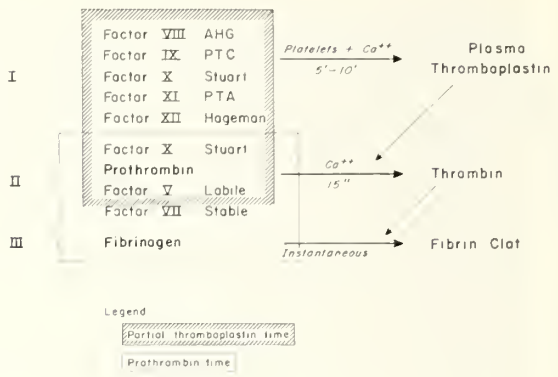


Fig. 2

the average time to complete each stage. We are primarily interested in the first Stage, the elaboration of thromboplastin, as it is in this Stage that congenital deficiencies of the hemophiliac factors result in abnormal amounts of thromboplastin and hence defective coagulation. The boxed, shaded area encloses all of those factors (and the hemophiliac factors are included) whose deficiencies are picked up by an abnormal partial thromboplastin time, PTT; and it is this test which has replaced the clotting time in the routine coagulation work-up.[5] The PTT is done exactly as the one-stage prothrombin time of Quick except that rather than add a form of thromboplastin to the patient's plasma, and by-pass Stage I, only cephalin, a source of platelets, is added thus reflecting both Stage I and Stage II of the intrinsic system. Normal values for the PTT is 40-100 seconds. Fig. 2 also makes clear that the combination of a P-time with the PTT covers all the coagulation factors. With a history, estimation of platelets, and tourniquet test these two tests would constitute the best coagulation work-up for the average patient.

The other test referred to as the specific test to determine which of the hemophiliac factors is involved is the thromboplastin generation test (TGT). The TGT is, however, a two-hour laboratory exercise for the most apt technician.

Finally, regarding therapy, until the day arrives when a daily supplement may be given to the hemophiliac as insulin is to the diabetic or Vitamin B12 to the patient with Addisonian anemia, we must concentrate our specific treatment to the bleeding episode. It is remarkable how much has been accomplished by the simple application of cold and pressure to a fresh injury and much more could be achieved if this simple principle could be spread to everyone entrusted with the care of a hemophiliac child.

I have mentioned already the basic sources of the specific hemophiliac principles; fresh blood or fresh frozen plasma for the classical hemophiliac and PTA or Factor XI deficiency, and stored refrigerated plasma for Christmas Disease. Limiting the problem of therapy to the classical hemophiliac as due to the lability of this factor and its very short half life, this is where the main

problem is; one must remember that to stop the various types of hemorrhage, minimal levels of 10-35% of AHG must be achieved and maintained for one to ten days. Fresh frozen plasma has on the average 65% of the AHG. The volumes of plasma to achieve this goal often causes circulatory overload.

Considerable progress has been made in England with large-scale production of concentrates of AHG from animal sources. The anti-genicity of this material preventing its use to one short course of treatment has discouraged most workers in this country. Interest here has been on the production of high potency concentrates of AHG. Commercially we have two preparations: lyophilized plasma and more recently AHG enriched fibrinogen.

I would like to mention a very bright star on the hemophiliac horizon regarding concentrates. When frozen plasma is thawed in the cold, a cold-insoluble precipitate clings to the plastic bag which contains considerable quantities of plasma AHG.[6,7] These concentrates have a potency (on a protein basis) thirty times greater than that of fresh frozen plasma. Any blood bank using the standard double plastic bag set who also possesses a refrigerated centrifuge can accomplish this in such a way that the concentrates can be taken from a unit of freshly donated blood, and that unit may still be used for routine blood replacement (deficient in AHG of course). The final product is potent, type specific, and inexpensive. The volume the precipitate is 3 cc and to it is added 10 cc of saline. Several of these concentrates can be put into a transfer bag and given at the proper time together. This technique is already in use in many larger hospitals. The concentrates have been prepared at the Maine Medical Center, although we have not used them in therapy as yet* One would envision a bank of these concentrates in a hospital as was previously done with fresh frozen plasma.

*Since the presentation of this paper, these cryoprecipitate concentrates have been used successfully several times.

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The Rheumatic Diseases

Laboratory Procedures and Diagnosis

ROBERT F. RITCHIE, M.D.*

Until recently laboratory confirmation for the diagnosis of a rheumatic syndrome was a "hit or miss" affair. Certain tests when positive offered support, but many patients with definite active disease had no serologic abnormalities. With the increase in the number and sensitivity of laboratory methods the percentage of seronegative patients with definite rheumatic disease has been markedly reduced. Consequently, the finding of negative serological studies make the presence of a rheumatic disorder unlikely. The purpose of this review is to summarize briefly current information concerning laboratory procedures that play a role in the diagnosis of the rheumatic patient.

General: The object of diagnostic laboratory methods is to demonstrate findings which: a.) indicate systemic disease, b.) suggest a particular group of disorders and c.) specify a discrete pathologic state. As investigators have evaluated each test and the moiety it measures, it has become evident that considerable skill and experience is required to properly evaluate the results. No test will yield "black and white" results and none should be interpreted as being diagnostic. Furthermore, no one test should be relied upon alone, but each should be interpreted in the light of complimentary procedures which measure the same or related factors. This is particularly true where difficult immunologic and serologic assays are concerned. Ideally, a single screening test should indicate the presence of disease within a particular system. Unfortunately, no such procedure is available and therefore, the clinician must employ a battery of tests which bracket all these disease states in the hopes of obtaining a diagnosis.

Nomenclature: Since the introduction of protein separation as a diagnostic tool, the nomenclature of protein types has been a source of confusion. In 1965 the World Health Organization met to establish an internationally acceptable terminology.[1] The immune globulins – those with the greatest number of synonyms – have been given the general term of Immunoglobulins abbreviated Ig. These comprise four major classes; IgG, IgA, IgM and IgD. Portions of the molecules have been identified as the K and L chains (Bence Jones proteins). Table 1 lists the immune globulin classes, physicochemical and physiological characteristics of each.

The term gamma globulin should be reserved for that portion of the serum proteins that migrate electrophoretically beneath the gamma region. As will be discussed below this is only a portion of the total immune globulins that was formerly designated "gamma globulins."

Sedimentation Rate: With the appearance of newer, more exotic, difficult and dramatic tests, the simple measurement of the erythrocyte sedimentation rate has lost its popularity. It remains, however, one of the most sensitive indicators of active systemic disease. The three methods that are in routine use, listed in the order of increasing sensitivity, are the techniques of Wintrobe,[2,3] Westergren,[3] and of Rourke-Ernstene.[4] These technically simple procedures measure the same plasma abnormality of increased fibrinogen and certain globulins.[5,6] The Rourke-Ernstene method measures the velocity of erythrocyte fall per minute while the techniques of Wintrobe and Westergren measure the distance fallen in one hour.

Falsely normal values that can be corrected by alteration of test conditions may arise. Cold agglutinin disease and cryoproteinemia are two disorders that may be encountered as possible rheumatic diagnostic problems. Erythrocyte sedimentation is prevented by the formation of a blood cell lattice in the former and a protein lattice in the latter giving rise to normal sedimentation rates at room temperature. The performance of the test at 37 degrees C will yield more correct values. Falsely normal values may also be encountered in patients with extreme elevation of blood viscosity due to large amounts of circulating macroglobulins (IgM) such as are often found in patients with Waldenstrom's Macroglobulinemia. Although no lattice is present as such, red blood cell fall may be inhibited by the viscous properties of the plasma. Manipulation of the test conditions cannot be practically performed to correct this error. Although Heparin is considered the best anticoagulant for the determination of the sedimentation rate,[7] EDTA is satisfactory. Should a normal sedimentation rate be obtained with EDTA when an elevation is expected, the use of other anticoagulants should be considered. The vast majority of rheumatic patients will have an elevated sedimentation rate often to a very high level.

Protein Electrophoresis: Electrophoretic separation of serum proteins perhaps more than any other laboratory test has caught the fancy of clinicians and researchers. Although a great deal has been written on the clinical significance of various electrophoretic findings only a very limited amount of information can be obtained. Because there are 5-7 peaks produced from human serum and at least 29 different protein species identified, several must migrate together to form some of the peaks. Although the albumin peak contains at least five species the great majority is albumin. The value for this most

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TABLE 1

SUMMARY OF THE CHARACTERISTICS OF THE IMMUNOGLOBULINS AS CLASSIFIED BY THE PRESENT SYSTEM OF NOMENCLATURE.

Immune-globulin	*Synonyms*	*Mwt. Sed. Value (Svedberg)*	*%Tot. Prot. gms.%*	*Function*	*Elevated in:*	*Depressed in:*
IgG	Gamma-2 Gamma ss 7S glob. 6.6S glob.	160,000 7S	15% 1.0-1.7 gms%	Antiviral & Anti-bacterial antibodies Prolonged immunity	Diffuse hypergammaglobulinemia IgG Myeloma Rheumatic Disease "Autoimmunity" Chronic Infection Liver disease Sarcoidosis Lymphoma, some Neophasia, some Malnutrition Burns Pyroglobulinemia Cryoglobulinemia	1° & 2° agammaglobulinemias Selective hypogammaglobulinemias Transient hypogammaglobulinemias of infancy Hypoproteinemic states IgA & IgD myeloma Macroglobulinemia
IgA	Gamma-1a Beta-2a	160,000 to 1 million 7-15S	3-4% 0.16-0.36 gms%	Anti-B subs. Skin Sensit. Present in ext. secret.	Diffuse hypergammaglobulinemia IgA Myeloma Rheumatoid Arthritis?	Ataxia-telangiectasia Selective hypogammaglobulinemias* IgG & IgD myeloma Macroglobulinemia
IgM	Gamma-1m Beta-2m 18S 19S	1 million 19S	2% 0.06-0.16 gms%	Acute phase immunity	Waldenstrom's Macroglobulinemia Mononucleosis Diffuse hypergammaglobulinemia Selective hypogammaglobulinemia Cryoglobulinemia	1° agammaglobulinemias Selective hypogammaglobulinemias Hypoproteinemic states
IgD	———	160,000 7S	<<1% 0.00-0.004 gms%	?	IgD Myeloma Selective hypogammaglobulinemia	?
K&L	Bence-Jones Light-chains	20,000-40,000 1-4S	Trace	—	Myelomatosis Macroglobulinemia "light chain" dis. Rheumatoid disorders Diffuse hypergammaglobulinemias	—
$\alpha',\beta,\gamma,\delta$	Heavy chains	50,000 4S	—	—	"Heavy Chain" disease	—

*IgA is found in the external secretions in normal concentrations

anodic peak, therefore, can be considered to be that of the true albumin content of the sample. At the cathode end of the strip the broad peak of "gamma globulin" is found which normally contains but a single protein-IgG immune globulin. In pathologic conditions, abnormal IgM and K and L chains can be found with a gamma mobility. A substantial portion of the total serum IgG content, however, is found beneath the alpha and beta peaks and therefore the value given for the gamma peak constitutes only a portion of the total immune globulins.

As has been pointed out by many observers, the finding of a low "gamma globulin" on paper or cellulose acetate electrophoresis is of little significance unless amounts of less than 300 mgs% are present.[8] This is particularly true in the pediatric age ranges. We have seen several children who have very low levels of serum "gamma globulin" on electrophoresis, however, when determinations of the total immune globulins were performed by more sophisticated techniques the values were found to be normal. The explanation is simply that significant amounts of the immune globulins moved electrophoretically with the alpha and beta globulins and were quantitated with those fractions. Elevations of the gamma peak can be of considerable clinical importance in the evaluation of the rheumatic disease suspect. Active disease is usually accompanied and may be preceded by elevation of the gamma peak and reduction in the size of the albumin fraction. Remission will usually be ac-

companied by return of these abnormalities towards normal. There are exceptions to this observation in the severely ill patient with systemic lupus, who terminally will show dropping values for the gamma peak.[9] This probably reflects increasing catabolism of the protein and a decrease in production.

Interpretation of the values given for the alpha and beta electrophoretic peaks is essentially impossible with the exception of those marked abnormalities seen in the dysproteinemias. The majority of the individual serum proteins migrate in the alpha and beta zones and few constitute a significant fraction of the total. Alpha$_2$ haptoglobin, the hemoglobin binding protein is elevated, occasionally massively, in chronic inflamatory conditions such as rheumatoid arthritis. The iron binding protein, transferrin constitutes the major portion of the beta$_1$ peak of cellulose acetate electrophoresis and is often found to be lowered in active rheumatic processes. The balance of the protein types are found in such small concentrations that no conclusion can be reached as to their pathological relationships.

To assist in the interpretation of standard electrophoretic methods the technique of immunoelectrophoresis should be considered.[10] Twenty-nine different proteins have been identified in human serum by this technique. Although the method is not difficult, interpretation of the results requires considerable experience.

The interpretation of electrophoretic values largely depends on the skills of the laboratory and the care with which its range of normals has been determined. Published norms may vary considerably for any given method and population[11] and should not be used to dictate the normal values for another laboratory.

Immunoprecipitation: Many researchers have become disenchanted with electrophoretic techniques for the quantitation of serum proteins. A possible future substitute is the determination of specific proteins by reaction with specific animal antisera. Several techniques are already available and yield accuracy considerably better than can be expected from electrophoresis.[12,13,14] Basically the methods involve the interaction of a single protein species with its animal antiserum. The result is a precipitate which can be developed in solution or in a support medium such as agar or cellulose acetate membranes. Quantitation of only six serum proteins have clinical significance as yet. They are: albumin, IgG, IgA, IgM, ceruloplasmin and B$_1$c compliment. Determination of only the first two can be obtained electrophoretically. The sensitivity of the immune precipitation techniques is so great that albumin and the gamma globulins can be easily measured in spinal fluid 200 times more dilute than serum. For the determination of CSF albumin only 0.05cc of fluid is needed and for IgG only 0.5 cc.

Tests For Rheumatoid Factors: The sera of many patients with rheumatoid arthritis contain antibody to the IgG immune globulin;[15] their own as well as that from other persons. The rheumatoid factor has been characterized as an immune globulin with a molecular weight of about one million, a sedimentation coefficient of 19S (Svedberg),[16] a structure made up of repetative units of smaller polypeptide chains, an electrophoretic mobility of beta-2, and has been given the designation of IgM (formerly gamma-lm, B2m globulin). This antibody complexed with IgG globulin circulates in the blood of rheumatoid patients as a macromolecule having a sedimentation coefficient of 22S (Svedberg).[17]

The currently available tests employ latex particles coated with normal IgG globulin. Commercially prepared reagents are in wide use, however, because of the considerable difficulties experienced with sensitivity and specificity, many institutions use the more accurate tube dilution method of Singer and Plotz.[18]

Although the rheumatoid factor is found commonly in patients with rheumatoid arthritis a protein indistinguishable from it is produced during the course of many other non-rheumatic diseases. Patients with subacute bacterial endocarditis frequently have a positive latex fixation test as do patients with hepatocellular disease.[19] Caplan has used the Eosin latex fixation test and has found that the method is a sensitive index of hepatocellular disorders and has used the test to screen asymptomatic blood donors for occult disease.[20] Patients with neoplasia or protein dyscrasias may also possess a positive latex fixation test which is persistant. Transient positives occur during the course of viral illnesses such as rubella. Macromolecules which have the properties of rheumatoid factors have also been described in certain pulmonary disorders in the absence of joint disease.[21] In all series reported, a small number of the normal controls have a weakly positive test. Conversely a certain number of patients with definite active rheumatoid arthritis have a negative test. Generally speaking rheumatoid patients have the greatest incidence of positive tests occasionally with titers as high as one and two million. Patients with nodular rheumatoid arthritis tend to have higher mean titers than those who do not.[22]

Several modifications of the procedures have been introduced. The most notable was the introduction by Hall which by concentrating the euglobulin fraction of serum could demonstrate a positive test in some sera that had previously been shown to be negative.[23] The euglobulin fraction contains the rheumatoid factors. A further increase in sensitivity is obtained by the inhibition technique of the same authors. In this modification the euglobulin fractions ability to interfere with a known positive serum is measured. These highly sensitive methods have their greatest value when results are completely negative, that is, when no rheumatoid factors can be demonstrated in the serum or euglobulin fraction.

Tests For The LE Factor: In 1949 Hargraves described a peculiar cell seen in the marrow preparation of patients with systemic lupus erythematosus.[24] Subsequently, this cell was also described in the peripheral blood of these patients. The LE cell phenomenon although known for the past 17 years is still unclear as to its mechanism.

The antibody involved has been characterized as a 7S, IgG globulin of 160,000 molecular weight[25,26] which can be absorbed from a serum by the addition of DNA.[27] There apparently is a close link between the LE cell and the extracellular material occasionally seen in the preparations.[27] Often, however, despite large numbers of extracellular bodies none can be found within polymorphonuclear leukocytes. These bodies both intra- and extracellular have been demonstrated to contain gamma globulin by fluorescent antibody techniques.[28] It would seem, therefore, that in addition to the factor for the production of the LE body another material is necessary for the ingestion of this nucleus-antibody structure. The number of LE cells has been correlated with the levels of "gamma globulin" but this can only be demonstrated in patients with SLE.[9,22] In the rheumatoid patient with a positive LE cell test no such correlation has been observed[22] suggesting that perhaps in the RA patient a different immune globulin is responsible for the phenomenon.

Because the LE prep requires the use of living polymorphonucleocytes the care with which the test is performed greatly effects the results. Although LE cells can be formed from intact living white cells, the mechanical disruption of some of the cells freeing nuclei increases the sensitivity. Sera that will react only with altered nuclei have been described.[29] In view of the fact that LE cells rarely exist in vivo the production of the typical cells *in vitro* requires a certain amount of time. Variations from the optimum incubation of one hour will introduce artifacts in the form of non-LE inclusions, poor cell morphology and in general make interpretation difficult. It has generally been agreed that for a cell bearing an inclusion to conform to the definition of an LE cell, certain criteria must be adhered to. The large round inclusion must be homogeneous and not vacuolated, must be stained the typical purple red color and be within an intact PMN. Anything short of this can only be suspect and should be evaluated by repeating investigation of the patients blood.

It should be kept in mind that the administration of cortisone or its analogs will alter the LE prep rapidly to make the cells atypical, difficult to interpret and may even eliminate them entirely.

The discovery of an LE cell in the blood of a patient should not in any way be interpreted to be diagnostic of SLE. Positives of various degrees have been described in up to 40% of rheumatoid patients 20% of patients with scleroderma, rubella arthritis, drug reactions of several types, polyarthritis and occasionally as we have recently observed in patients with only vague musculoskeletal complaints and as yet no definable disease. Patients with certain forms of liver[30,31] disease and those with leprosy[32] have also been observed to possess the phenomenon. The number of cells observed on a smear is valuable information and should be included in the laboratory report. Although it is at best a semi-quantitative determination the finding of a "rare" cell verses a "loaded" prep have greatly different significance. The latter being much more suggestive of active SLE or one of the "overlap" syndromes.

With the knowledge that the LE factor is probably an anti-DNA antibody, at least in those patients with definite active disease, commercial firms have prepared slide tests in the hopes of offering a simple substitute for the LE prep. These methods employ DNA-nucleoprotein coated latex particle which when mixed with the serum of certain LE patients will agglutinate. The sensitivity and specificity of the techniques are such that the conventional LE prep is in no danger of replacement.

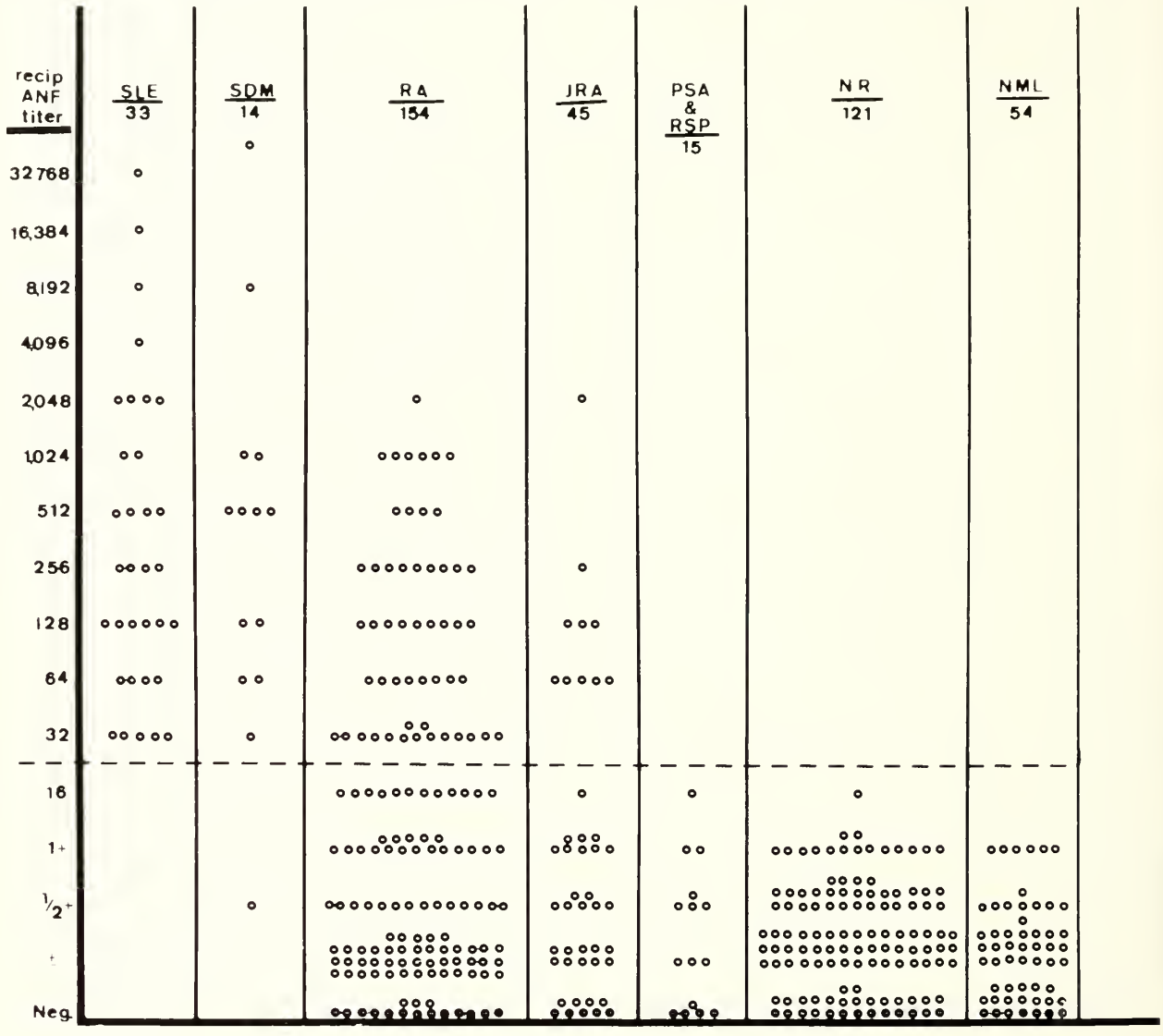
Sophisticated techniques for the demonstration of antibody to nuclear fractions are available in the DNA-Bentonite[33] and the nucleoprotein coated latex particle[34] techniques. Both methods are sensitive and are not difficult to perform. A more complex test measures the ability of the anti-DNA-DNA reaction to utilize compliment.[35]

Tests For Antinuclear Factors: Closely allied but not identical to the LE factor is a large group of immunoglobulins that react with constituents of the mammalian nucleus, the antinuclear factors (ANF). This group contains antibodies that bind to nuclei in five major patterns classified descriptively as: homogeneous, speckled, peripheral, shaggy and nucleolar.[36] Each indicates that the globulin reacts with a different portion of the nucleus and is identified by the distribution of fluorescent tagged antibody when observed under the U-V microscope. Further subdivision of the factors are made by classifying them according to their molecular type, ie. IgG, IgA or IgM. Depending on the technique used, a high percentage of patients with rheumatic diseases will have one or more of these antibodies in there sera.[9,22,37,38,39] In general patients with SLE are positive in virtually every instance as are cases of scleroderma. Rheumatoid patients are positive in a great proportion of instances but in only one third will a strong positive be found.[22,35] The lowest incidence is found in the sera of patients with either periarteritis nodosa or some other form of vasculitis not primarily diagnosed as one of the definite rheumatic syndromes.

There are several forms of the test in use today. The greatest difference is in the material used as the antigen. Most methods use frozen tissue sections, WBC or buccal mucosal cells. The method described by Friou[40] employs spots of nuclear extracts which contain either DNA or nucleoproteins fixed on to microscope slides. The tissue sections may be of calf thymus, rat liver or spleen, human tissues such as liver spleen, skin, placenta[37] etc. Work done by us has shown that the more metabolically active the tissue, the greater the sensitivity obtained. A tissue of relatively constant antigenic content and activity is obtained from young laboratory rat liver. When frozen sections are made from this tissue and fixed in an acetone-alcohol fixative excellent sensitivity, reproducability and morphology is obtained.[22]

From a large group of patients with both rheumatic

TABLE 2
DISTRIBUTION OF THE "HOMOGENEOUS" TYPE OF ANTINUCLEAR FACTOR IN VARIOUS DISEASES AND IN NORMAL CONTROLS. THE DASHED LINE INDICATES THE UPPER LIMITS OF NORMAL.



and non-rheumatic disorders tested by a sensitive technique it was established that a substantial number of persons without obvious disease, ie "normals," had detectable quantities of "immune globulin" that reacted with nuclei[22] (Table 2). The difference between these persons and those who were positive but had rheumatic disease was that the titers rarely exceeded 1:8 and were never higher than 1:16. On the other hand all patients with SLE were above 1:32. The titer of 1:16 was therefore chosen as the upper limits of normal. Values below this were not considered to be of diagnostic significance.

In examining the sera of patients with minimal musculoskeletal complaints it has been found that a small number had significant quantities of ANF. The answer as to why these factors are present in apparently normal people is not yet apparent.

Although many investigators have found persons in the control groups to have a positive ANF test it is probably not safe to interpret them as being false positives. There are a group of patients, however, who do have a false negative test. On examining a large number of sera 13 were found who would have been called either negative or weakly positive if only the whole serum had been examined.[22] Because the method employed in this laboratory examines the whole serum and a dilution of 1:8 the observation was made that these patients were considerably more positive in the diluted sample. Further investigation indicated that titers were often of a very high level and clinical review indicated

TABLE 3

SUMMARY OF TEST PROCEDURES PERTINENT TO THE RHEUMATIC DISEASES.

Test	*Protein Measured*	*Type of Immunoglob. Measured*	*Disease Specificity*	*Sensitivity*	*Rheum. Disease Positive*	*Other Positives*	*False Values*
Sedimentation Rate	Fibrinogen & some globulins	?	Non-specific	High	Increased when active	Most systemic diseases	Cold agglutinens Cryorpoteinemia High viscosity syndrome
Serum Electrophoresis	Serum proteins Alb,α_1, α_2,β,γ	Partial IgG	Non-specific	Mod.	Increased gamma when active*	Allergy Sarcoid Vasculitis Chronic Inf. Dysproteinemia Amyloid Liver disease	Gamma globulin measured as α and β globulins
Immune precipitation	Specific serum proteins	IgG,IgA,IgM	Non-specific	High	Increased IgG*	All above Mononucleosis (IgM)	
Latex Fixation	Rheumatoid Factor	IgM	Mod. specific	High	Variable with disease	Hepatocellular disease SBE Viral diseases Rubella arthritis Normals (3%) Neoplasia	aged or improperly stored reagents
LE preparation	LE Factor	IgG	specific	Mod.	Variable with disease	"Drug" LE Hepatitis, Lupoid Leprosy Rubella arthritis	Artifacts from prolonged incubation
Antinuclear Factors	Anti-nucleoprotein Anti-DNA Anti-nucleolar	IgG,IgA,IgM	Mod. specific	High	Variable with disease	"Drug" LE Hepatitis, lupoid Leprosy Rubella arthritis Liver disease Raynaud's phenom. Keratoconjuctivitis sicca Thyroiditis Normals (50% low titer)	"False negatives" from steroids

*Serum "gamma" globulin may be depressed terminally in SLE[9].

that these were all rheumatoid patients who were receiving parenteral costicosteroids. The nature of this interesting phenomenon and its relationship to steroids is currently under investigation.

DISCUSSION

Rheumatic disease, often mimicing other more clearly defined syndromes, may present in a wide variety of clinical forms. Nonspecificity of historical and physical findings is the rule in this confluent group of diseases. The laboratory data reflects this overlap. Although it may often be an exercise in academics to classify rheumatic patients, it has practical importance when a therapeutic decision must be made. Patients are frequently seen who when carefully evaluated, have characteristics of more than one clinical rheumatic syndrome. A dilemma is met when a therapeutic regimen may be suggested on one hand and contraindicated on the other. This is exemplified in the all to common patient who presents with findings of both scleroderma and SLE or SLE and rheumatoid arthritis. In either instance steroids may be indicated for the patient with SLE but would be contraindicated for those with scleroderma and inadvisable for those with rheumatoid arthritis. Similarly, it would seem that the diagnostic differentiation between rheumatoid arthritis and osteoarthritis would not present a problem but, on occasion quite the reverse might be the case. The laboratory will add another parameter which

TABLE 4

SUMMARY OF TEST PROCEDURES AS RELATED TO THE VARIOUS RHEUMATIC DISEASES.

TEST	SLE		SCLERODERMA		RA		JRA		GOUT	
	Active	Inactive	Active	Inactive	Active	Inactive	Active	Inactive	Active	Inactive
Sedimentation Rate	↑↑↑↑	N-↑	↑↑↑↑	↑	↑↑↑↑	N	↑↑↑↑	N	↑-↑↑↑	N
Serum Electrophoresis										
-Albumin	↓↓ *	N	↓↓	↓	↓↓	N	↓↓	N	↓	N
-Gamma glob.	↑↑↑↑	↑-N	↑↑↑↑	↑↑-N	↑↑-N	↑-N	↑↑-N	↑-N	N	N
Immune-Precipitation										
-IgG	↑↑↑↑	↑-N	↑↑↑	↑	↑↑↑	↑-N	↑↑	N	N	N
-IgA	Var.	Var.	Var.	Var.	↑	Var.	Var.	Var.	Var.	Var.
-IgM	↓-N	N			↑-N	↑-N	N	N	N	N
Latex Fixation	20% ↑	20% ↑	20% ↑	20% ↑	60-85% ↑	less	20% ↑	less than 3%	less than 3%	Less than 3%
LE Preparation										
LE cells	60-100% ↑	Much less	20-50% ↑	Less	40% ↑	0	Occ. ↑	0	0	0
ECM	↑	↑	↑	Less	↑↑	↑	Occ. ↑	0	0	0
Antinuclear Factors										
Homogeneous	↑	↑↑	0-↑↑	0-↑	0-↑↑↑	0-↑↑↑	0-↑↑	0	0	0
Speckled	↑↑↑	0-↑	↑↑↑	↑↑↑	0-x	0	0	0	0	0
Peripheral	↑↑↑	0	0	0	x	0	0	0	0	0
Nucleolar	↑↑↑	↑	↑	↑	↑	0	0	0	0	0

↑ = elevation or positive ↑↑↑↑ = marked elevation N = Normal ↓ = depressed

*Serum gamma globulin levels may be depressed terminally[9]

will lend support to a diagnostic impression and the therapeutic program indicated.

In addition to the diagnostic assistance offered by rheumatic disease-oriented tests there is accumulating evidence that certain serologic findings will indicate those arthritics who will pursue a difficult course and who will need close surveillance. A British study[41] has suggested that some rheumatoid arthritics have certain serologic abnormalities very early in their disease – a high titer of ANF – and will suffer progressive joint destruction and have a shortened life span. Furthermore, studies in this laboratory indicate that some patients may have serologic findings prior to the onset of clinical disease and it is in this group that the role of drugs may be an important factor in the precipitation of overt disease. The list of medications that are implicated in the "unmasking" of systemic lupus or a lupus-like syndrome continues to grow and because evidence is available that serologic abnormalities may be present prior to the onset of symptoms, the search for these factors is more than academic. Obviously, to screen every patient about to receive a suspected medication is impractical, however, when the physicians evaluation includes findings such as arthritis, Raynaud's phenomenon, morning stiffness, repeated pleurisy, hematuria, etc., a laboratory study might avert a serious iatrogenic complication. The suspicion that a drug is capable of precipitating an illness should in no way detract from its value in the majority of patients. What is suggested, however, is that the physician should continually be reminded that for certain patients these drugs have disease potential of themselves.

For the reason that at present no single screening test for rheumatic disease is available, a battery of procedures must be performed. An individual test is of little value and its evaluation must be in the light of complimentary tests. The "battery" should include a sedimentation rate, LE prep, ANF test and a latex fixation test at a minimum. Measurement of total protein, and protein electrophoresis (or quantitative immune globulin assay) will increase the value of the battery.

To simplify the complex interrelationships of the laboratory findings, Tables 3 and 4 summarize expected results in the majority of cases. The reader must remember that for an individual case exceptions will occur and must be anticipated.

Summary

A brief description of the tests pertinent to the evaluation of the rheumatic diseases has been presented with a review of the current system of nomenclature for the serum proteins involved in the immune process. It is stressed that evaluation of the rheumatic patient cannot be complete without appropriate laboratory investigation. Also suggested from recent information is that patients with minimal complaints may have severly disturbed serologies indicative of a subclinical rheumatic syndrome which may have great implication in the prevention of iatrogenically precipitated disease.

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Factors in the Choice of an Internship

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What attracts interns to one hospital and not to another? The type of hospital? The size of the hospital? The location of the hospital? The type of internship offered? The housing accommodations available? The size of the stipend? The education program? The amount of patient care responsibility granted to the intern? The opportunities for future residency or future practice? The percentage of autopsies obtained in the hospital? Each candidate considers several of these factors, but no one candidate considers all of them.

The material in this report was collected by utilizing a form questionnaire and from personal correspondence with candidates who applied to us, but chose another hospital for their internship. By correlating the information, thus obtained with the candidate's application file and interview data, an attempt has been made to evaluate the factors leading to a final choice of internship from the point of view of the candidate.

Before examining the data, it is necessary to establish a frame of reference for the reader. It is useful to have background information about the hospital (the Maine Medical Center), the candidate (those assigned to the hospital as well as those who chose another hospital) and the methods of collecting the data.

THE HOSPITAL

The Maine Medical Center is a 400 bed voluntary, non-profit Community Hospital in Portland, Maine. It is one of several hospitals serving Maine's area of greatest population density and it is the state's largest hospital. More than 25% of the patients admitted come from outside the immediate Portland area.

The hospital, like most community hospitals, was established for one purpose: to provide patient care. Although education and research may have been included among the hospital's original objectives, they existed in word, not in fact. More than a decade ago the Maine Medical Center took the first step to accept education and research as fundamental components of quality patient care. Since Maine does not have a medical school, it was felt a strengthened intern and resident program was necessary as a key building block in the hospital's education programs. The hospital has had house officers since 1892. Though formal approval did not exist, internships were developed at the hospital more or less since 1899. Residencies in internal medicine and general surgery were established shortly after WW II. Subsequently, residencies were established in anesthesia, general practice, pathology, pediatrics and radiology. To coordinate and give direction to house staff training activities, a full-time director of medical education was employed in 1960. In recent years, the hospital has been listed as a major affiliate hospital of the Tufts University School of Medicine.

TABLE 1

MEDICAL SCHOOLS AND STATES OF ORIGIN OF INTERNS AT THE MAINE MEDICAL CENTER 1960-66

Medical School		*State or Country of Origin*	
Boston University	3	Connecticut	5
Bowman Gray	1	Georgia	1
Buffalo	2	Illinois	2
California, University (S.F.)	1	Maine	25
Colorado	1	Massachusetts	13
Columbia	1	Michigan	2
Emory	1	Mississippi	1
Hahnemann	3	New Hampshire	9
Harvard	1	New Jersey	3
Illinois	1	New York	7
Loma Linda	1	Ohio	5
Marquette	1	Pennsylvania	7
McGill	1	Rhode Island	2
Medical College of Virginia	1	Washington	1
Michigan	3		—
New York Medical College	1	U.S.A.	83
Ohio	3	Korea	1
Pennsylvania	3		—
Pittsburgh	1		84
Temple	5		
Tufts	38		
Vanderbilt	1	26 Medical Schools	
Vermont	7	14 States	
Washington University	1	1 Country outside U.S.A.	
Yale	1		
Seoul, Korea	1		
	84		

The hospital participates in the National Intern Matching Program which operates annually from May through March of the following year, when intern appointments are announced for the current year. The hospital begins to receive correspondence and meet candidates for interviews during the latter part of the candidate's third year. Correspondence and visits by students increase and reach a peak during October and November. A few candidates are seen as late as January, the month applications close.

The number of inquiries varies from year to year between 150 and 200. Inquiries are received from students enrolled in 25-35 United States and Canadian Medical Schools regarding our 12 internships. The past two years has seen a marked increase in the number of inquiries from foreign medical schools.

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TABLE 2

INTERNS AT THE MAINE MEDICAL CENTER 1965-66

1965			1966		
Premedical School	*Medical School*	*State of Origin*	*Premedical School*	*Medical School*	*State of Origin*
Allegheny	Boston University	Connecticut	Bowdoin (3)	Hahnemann	Illinois
Amherst	Columbia	Georgia	Bowling Green	Illinois	Maine (3)
Bates	Emory	Illinois	Franklin and Marshall	Ohio	Massachusetts (2)
Bucknell	Hahnemann	Maine	Haverford	Pennsylvania	Ohio (2)
Dartmouth	Marquette	New Hampshire	Swarthmore	Tufts	Pennsylvania (2)
Emory	McGill	New Jersey	Rutgers	University of Washington	Washington
Lehigh	Ohio State	New York	Wisconsin		
Maine	Pennsylvania	Ohio (3)	Yale	Seoul	Korea
Marquette	Temple	Pennsylvania	Seoul, Korea		
Ohio State (2)					
Williams					
9 Medical Schools			7 Medical Schools		
9 States			6 States and Korea		

THE "LOOKER" AND THE "EARNEST CANDIDATE"

Only those students who complete an application are considered candidates for the purposes of this report. They fall into two groups: the "looker" and the "earnest candidate." The "looker" is apt to view the hospital much as a curiosity seeker without apparent serious intention of taking an internship at the hospital. The "earnest candidate" on the other hand gives the internship thoughtful consideration. It is often difficult to categorize a candidate, and at times the "looker" cannot be separated from the "earnest candidate" until after the results of the National Intern Matching Program are announced.

The hospital and its training program sooner or later develop a reputation and this reputation is often influenced by courtesies shown to former "lookers." Candidates are attracted to visit a program by a suggestion made to them by upper classmen who were previous "lookers." The "lookers" of one year have been the known source of candidate referrals for interns two or three years later. Repeated instances have occurred where highly desirable candidates were referred by a "looker" who lived across the hall in the same dormitory or served an internship in the same hospital.

Complete data is not available on class standing of candidates. The data available suggests that candidates at this hospital come from the upper and the middle thirds of the medical school class. To be sure there are extremes, and each year both the academically successful and the academically less successful apply to the program. So far as can be determined, the academic standing of those who enter the internship and those who take a program in another hospital is nearly the same. It is our impression from candidates' files that more of the high ranking students tend to follow academic or research careers in medicine and are sought by the medical school hospital.

THE EARNEST CANDIDATE

To understand the intern applicant it is necessary to know both about the intern in the program as well as those who accept appointments in another hospital. Complete data is available on the interns who served their internship at this hospital from 1960 through 1966 (Tables 1 and 2). In the seven years this hospital obtained the full complement of interns under the National Intern Matching Program.

During the years 1960-66 interns came from 26 medical schools. Nearly one-half (38) from Tufts University School of Medicine, and 7 from the University of Vermont School of Medicine. Candidates came from 14 states and Korea. (One graduate from a medical school outside the United States was rated sufficiently high in the 1966 matching program to be assigned to the program.) The state of origin is designated as the state from which the candidate entered medical school. Maine leads the list with 25 interns followed by other states in the Northeastern part of the nation. A total of 54 candidates came from the New England States and 22 from New York, New Jersey, Pennsylvania and Ohio.

For a number of years it has been generally accepted that intern candidates came to the Maine Medical Center because they either attended summer camp or college in Maine. A review of the application data suggests that this is true in part, but it is not as important a factor as to be a major source of candidates.

The location of the medical school shows the greatest number to come from New England, the Middle Atlantic and the North Central states with a scattering of schools over the nation. Again Tufts and Vermont lead the list.

For those candidates who accepted an internship in another program, complete data is available only for the years 1965-66. Table 3 summarizes the pertinent data.

In 1965, 27 replies were received from 29 question-

TABLE 3

INTERN CANDIDATES ACCEPTING HOSPITAL APPOINTMENT OTHER THAN MAINE MEDICAL CENTER 1965-66

1965				1966			
Medical School		*State*		*Medical School*		*State*	
Colorado	2	Connecticut	3	Albany	1	Connecticut	1
Duke	1	Illinois	1	California (S.F.)	1	Washington, D.C.	1
McGill	1	Maine	2	Cincinnati	1	Hawaii	1
New York Medical College	1	Massachusetts	8	Harvard	1	Illinois	1
		Minnesota	1	Howard	1	Maine	3
Ohio	1	Montana	1	Jefferson	1	Massachusetts	5
Pennsylvania	1	New Jersey	2	Kansas	1	New Jersey	1
Pittsburgh	1	New York	3	McGill	1	New York	1
Rochester	1	Ohio	1	Medical College of		Ohio	1
Stritch	1	Pennsylvania	1	Virginia	2	Pennsylvania	1
Syracuse	1	Rhode Island	2	New York Medical College	1	Virginia	2
Temple	3	Texas	1	Pennsylvania	1	Washington	1
Tufts	10	Washington, D.C.	1	Tufts	5	Wisconsin	1
Vermont	1		—	Vermont	2		—
Washington University	1		27	Wisconsin	1		20
Yale	1			Woman's	1	Canada	1
	—				—		—
	27				21		21

naires (93%). Candidates came from 15 medical schools and from 13 states. For the year 1966, 21 replies were received from 24 questionnaires (87%). These candidates came from 15 medical schools and 13 states and Canada.

THE QUESTIONNAIRE

The questionnaire (reproduced in Table 4) was designed to examine the impressions of candidates who chose an intern program elsewhere. It deals with subjective responses based on value judgments which are difficult to interpret as statistical data. Even so, it was thought the information might assist in determining strengths and weaknesses of this hospital's training program. If so, the material may be useful to training programs in other community hospitals.

A study of the questionnaires occasionally reveals a startling observation or two when candidates point out that our training objectives are not always consistent with current objectives as seen by the intern candidate. It is obvious that hospitals will not long maintain a house staff training program unless interns and residents are present. House staff will not accept a training program which does not appeal to them.

Often valuable information is derived from comments and personal letters which are returned with the questionnaire. The letters detail observations made by the candidate. Many of these observations are surprisingly accurate and pinpoint known strengths and weaknesses of the program. The questionnaire serves as a springboard for the observation.

DISCUSSION

Reference to the data tabulated from the questionnaires suggests findings of interest. It will be noted that 26 of the 48 candidates took appointments at another community hospital. About half (14) took appointments in hospitals with a medical school affiliation; 11 in hospitals without a medical school affiliation.

The size of the hospital appeared to be a factor in the choice of internship, but the data did not indicate the importance of this factor. Two candidates chose hospitals of less than 300 beds. Twenty-one of the 48 replies indicated the candidates selected hospitals of 300-500 beds.

Twenty-four candidates selected hospitals in New England, 13 in the Middle Atlantic States, thus 37 of the 48 candidates took appointments in hospitals in the Northeastern part of the country. This data is consistent with the fact that most of the candidates who consider this hospital's program come from medical schools in the Eastern part of the nation.

Thirty-two of the 48 candidates, a clear majority, took rotating internships. By itself, this number is probably meaningless since candidates originally intended to focus on a rotating program.

Of the hospitals chosen, about one-half of the candidates selected hospitals which offered housing or subsidized housing for married house staff.

Stipends alone do not attract interns, but adequate stipends for house staff are necessary. Generally speaking, budgetary restrictions make it impossible to attain the ideal house staff stipend.

The quality of the educational program remains one important factor in the choice of internship. The results of this question regarding educational programs were difficult to interpret since answers require a value judgment. Forty-four of the 48 candidates either considered the Maine Medical Center program good or excellent. Yet, each individual defines a good and excellent intern-

TABLE 4

TABULATED RESULTS OF QUESTIONNAIRE 1965-1966

	1965	1966	Tot.
1. What type of hospital did you select as your first choice?			
A community hospital	12	14	26
Affiliated with a medical school	9	6	15
Not affiliated with a medical school	5	6	11
A state or municipal (charity) hospital	2	3	5
A medical school hospital	7	4	11
A federal hospital (Army, Navy, Public Health Service)	4	2	6
2. Size of hospital			
a. Was the size of the hospital important in your choice?			
Important	19	13	32
Not important	8	8	16
b. What size hospital did you list as your first choice?			
Less than 300 beds	2	0	2
More than 300 beds; less than 500 beds	12	9	21
More than 500 beds; less than 700 beds	4	6	10
More than 700 beds	9	6	15
3. Type of internship chosen by candidate			
Rotating	19	13	32
Mixed[a]	5	5	10
Straight[b]	3	3	6
4. Location of hospital of your first choice			
New England	14	10	24
Middle Atlantic	6	7	13
South	0	0	0
Midwest	3	3	6
Mountain	2	0	2
Pacific	2	0	2
South West	0	1	1
5. Housing Accommodations			
For single house staff	23	18	41
For married house staff	11	16	27
Both of above	11	8	19
Must seek own housing	7	3	10
6. Stipend			
a. Was the amount of stipend important in your choice of hospitals			
Yes	12	9	21
No	15	12	27
b. Was the stipend offered at the hospital			
Too little	6	3	9
Satisfactory	21	15	36
Don't know	0	3	3
7. Educational Program			
a. Was the educational program the primary factor in your choice of internship?			
Yes	14	18	32
No	10	6	16
b. From the information you were able to obtain by mail and interview and from what you now know you would rate the internship at this hospital as			
Excellent	11	7	18
Good	15	11	26
Average	1	1	2
Fair	0	0	0
Poor	0	0	0
Don't know		2	2
8. Patient responsibility			
a. Was patient responsibility important in your choice of hospital?			
Yes	22	14	36
No	5	7	12
b. Amount of patient responsibility given to intern staff at hospital			
Sufficient	24	18	42
Insufficient	0	0	0
Don't know	3	3	6
9. Future residency: Was the opportunity for future residency important to you?			
Yes	19	18	37
No	8	3	11
10. Future Practice			
a. Were opportunities to practice in this area in your choice of hospital important?			
Yes	13	9	22
No	14	12	26
b. Did you consider them adequate in Maine?			
Yes	16	15	31
No	4	1	5
11. Autopsy Data			
a. Were autopsy data important in your choice of internship?			
Yes	11	2	13
No	16	19	35
b. The autopsy data of the inquiring hospital were:			
Too low	0	0	0
Satisfactory	15	9	24
Of little or no concern	8	2	10
Don't know	5	9	14
12. Personal plans upon completing your inquiry internship			
a. Enter general practice?	6	3	9
Immediately	1	0	1
After further training	2	6	8
b. Further residency training?[c]	22	16	38
c. Ultimate plan to specialize?[d]	14	10	24
d. Plans not completely formulated	5	3	8

[a]Medical 9; Surgical 1

[b]Surgical 2; Medical 2; Pathology 1; Pediatrics 1

[c]Pediatrics 3; Surgical 9; Ear, Nose, Throat 3; Medical 12; Orthopedic 1; Psychology 4; Obstetric Gynecology 4; Anesthesia 1 Radiology 1

[d]Pediatrics 3; Ear, Nose, Throat 3; Orthopedics 1; Surgical 8; Psychology 4; Obstetric Gynecology 3; Medical 6; Anesthesia 1

ship differently. It is obvious that patient responsibility remains a dominant factor in a "good" internship.

Thirty-seven of the 48 candidates indicated that they did not choose their internship at the Maine Medical Center because of restricted openings for residents. It is apparent that a lack of sufficient number of training programs or a sufficient number of openings in available programs was important. At the same time, the future practice opportunities were not as important consideration as some physician advisors would indicate. In the eyes of the candidate surveyed, they were adequate in Maine.

It is interesting to note that while the Council on Medical Education of the American Medical Association insists upon adequate autopsy percentages, the candidates do not stress the importance of these statistics. In our study, some importance was attached to the autopsy percentages in 1965 but less so in 1966, when only 2 indicated that autopsy data was considered.

One of the most valuable contributions revealed by the questionnaire was derived from the comments and personal correspondence with the candidates. Reasons for not choosing the Maine Medical Center program were classified into three groups: (1) characteristics of Maine, such as geographic, political, economic, and culture of the area (2) the hospital (including the house staff training program) (3) personal reasons. The first group included reservations such as a geographic location out of the main stream of professional activity. This reservation was further emphasized as important because there is not a medical school in the immediate area. Accordingly, some candidates see this as a lessened opportunity for the practicing physician to find the stimulation of academic medicine. Others indicated the area lacked educational opportunities outside of medicine for the physician and his family. Included in the group were impressions of the lack of opportunity for cultural, and educational stimulation afforded in the very large city. Although a number of sport enthusiasts candidates wished to live in Maine because of the long winters and the abundance of snow; others considered the weather as a disadvantage. Among the objections noted in this group included those which border on physician training. Hospitals involved in an active house staff training program should have broadly based resident opportunities. For example, residencies should be offered in all specialties of medicine which might be of interest to the candidate. Failure to provide residency training in most specialties requires an intern to move to one community for an internship and to another community for residency training. Today, since most interns are married, the added move places an additional financial burden on the candidate.

The second group, the hospital group, was divided into two subgroups: general and educational objections. Among the general was a comment about hospital size. A hospital of less than 500 beds, in the eyes of some candidates, was too small for complete internship training. Though a hospital may maintain a medical school affiliation, it should be in close proximity to the medical school. A medical school affiliation should be direct, obvious, and formally defined. Some objected to the fact that while a hospital has a program of basic clinical research, the research department should not be restricted to certain areas of investigation. It should be a fairly large department. Lastly, among the general objections was included the fact that the physical facilities of a hospital cannot be old and outdated and there must be provision for housing or subsidized housing for married house staff.

The second group of hospital objections included shortcomings in educational programs as observed by the candidates. Hospitals which offer only a rotating internship should consider offering straight or mixed programs. Some believe that private patients should be more extensively utilized in teaching. Ideally, the candidates thought there should be a combination of private and service patients used in the teaching.

A night and weekend assignment for interns which operates on an every other night and every other weekend basis was labelled by some as an excessive workload. It is a schedule which "wrecks family life and amounts to slave labor." Others commented that an internship may be unusually good, but there must be a "wide range for resident training." Another comment on stipend noted that while a stipend may be adequate for single house officers, it must also be adequate for the married house staff. Many hospitals provide adequate stipends for single, but not married house staff.

Third, the personal reasons included items such as, lack of opportunity to visit the hospital. Because the candidate wanted some personal contact with the hospital before accepting an appointment, he did not wish to elect a program with which he was not personally familiar. Because of the great travel distance, he was unable to see the hospital. Others mentioned that since the hospital failed to offer straight and mixed programs, the internship did not meet their needs. Occasionally, candidates were seen in which there was a breakdown in communications between the hospital representative and the candidate. These individuals indicated that the hospital did not positively assure them that they were offered an appointment. They were left with the feeling that the hospital was not interested in them as a candidate. As a result, they did not consider an appointment at the hospital. They added that other hospitals greeted the candidates with considerable interest and assured them of an appointment.

Conclusions

1. Interns tend to take their training in medium sized (300-500 beds) or larger (more than 500 beds) hospitals.

2. Intern applicants to one community hospital are more likely to come from both medical schools and states within the hospital region, but the area of the "region" is larger than formerly believed.

3. A majority of the intern candidates interviewed and applying at this hospital are looking for a good rotating internship because

   a. They want an internship which provides them with a broad background for subsequent community service as a family practitioner or as a physician providing first-line medical care.
   b. Many fourth year medical students remain undecided about their choice of specialty and they want more time and clinical exposure to weigh their decision.

4. House staff stipends are not an important element in the choice of internship in the group surveyed.

5. Housing for married house staff supported or subsidized by the hospital, appears to be a more important factor in the intern's choice of a hospital than is the stipend.

6. In attracting interns, the importance of a hospital-medical school relationship (affiliation) cannot be overstressed. There is a need for this relationship to be direct, obvious, and generally known.

7. The intern's potential opportunity for obtaining a residency in the hospital of his internship, (either through a wide variety of residencies or through a sufficient number of openings in each residency) is one paramount issue in the choice of an internship.

8. The percentage of autopsies in the hospital is given little or no consideration in their choice by intern candidates.

9. Interns choose another hospital because of:

a. Geographic, educational, or cultural reasons

b. Hospital features (including the training program)

c. Personal considerations

SUMMARY

Patient care in the modern hospital is appraised by numerous techniques. Hospitals with house staff education should be examined similarly. One method whereby the educational program may be appraised is to examine some of the parameters of its image as seen by the intern applicant.

The impressions of nine years observations (two in one hospital; seven in another) and the detailed results of years 1965 and 1966 are presented. The studies were made using a simple questionnaire together with the candidate's application file (including formal application and supporting letters from the medical school dean and others) as well as impressions formulated during personal interviews.

Institute on Team Work for the Chronically Ill

September 21-23, 1966

Banquet Session — September 22, 1966

The Maine Department of Health and Welfare will sponsor an Institute on Team Work for the Chronically Ill at the Shawmut Inn, Kennebunkport, Maine on September 21-23, 1966.

Of special interest to physicians will be a banquet session at 6:30 p.m. on September 22nd at the Shawmut Inn. The Chairman of this session on "Medicine and Team Work" will be Daniel F. Hanley, M.D., Executive Director of the Maine Medical Association and Editor of The Journal of the Maine Medical Association. Ellsworth T. Newmann, M.D., Administrator, Massachusetts General Hospital, Boston, will be the speaker.

Changes in Program

The August issue of the Journal published an outline of topics and speakers for the three-day Institute. Please note the following changes:

On September 21st Ezra Saul, Ph.D., Professor of Psychology, Tufts University, and American Hospital Association fellow at Tufts — New England Medical Center, Boston will speak on "Communication and the Team" in place of Sol Levine, Ph.D.

On September 23rd John Lorentz, M.D., Director, Department of Medical Rehabilitation, Maine Medical Center, Portland will be the Chairman and Moderator of "Team Work — Rehabilitation Services in a Maine Hospital." Team members will include: **Medical Social Work:** Gwendolyn Norton, Social Worker, Social Service Department, Maine Medical Center, Portland; **Physical Therapy:** Rowena Nelson, Chief Physical Therapist, Maine Medical Center, Portland; and **Occupational Therapy:** Patricia Robinson, Occupational Therapist, Maine Medical Center, Portland.

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- **Ileostomy**
- **Gastroenteritis and colitis**

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1-2 years	5 mg.	½ tsp. 5 times daily
2-5 years	6 mg.	1 tsp. 3 times daily
5-8 years	8 mg.	1 tsp. 4 times daily
8-12 years	10 mg.	1 tsp. 5 times daily

Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily) Based on 4 cc. per teaspoonful. Maintenance dosage may be as low as one-fourth the initial daily dose.

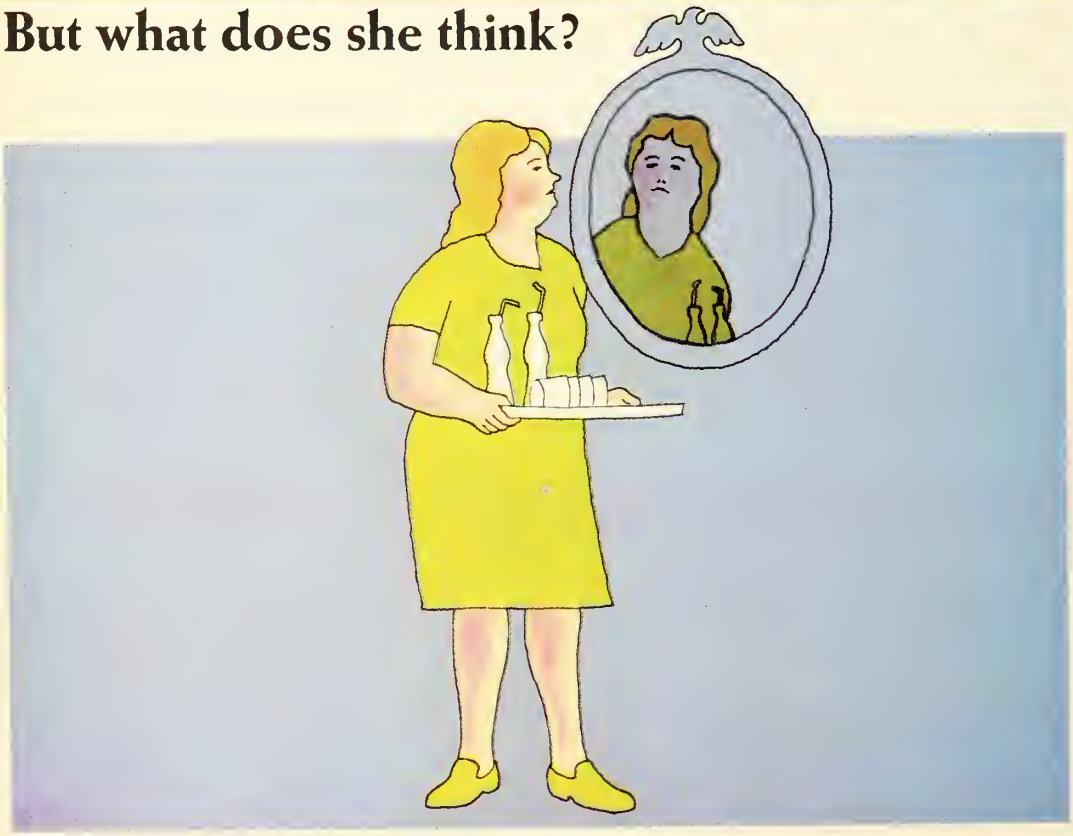
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Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneuratics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring alertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libido, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Radiation Therapy of Lung Cancer

Maine Medical Center, 1955-1965

ROBERT P. ANDREWS, M.D.* AND ROBERT A. BEAROR, M.D.**

INTRODUCTION

The results of palliative treatment of lung cancer in 141 patients at the Maine Medical Center in Portland in the past ten years have been reviewed and are presented here. The number of patients seen annually during that period has doubled and in the months since completion of this statistical review in June, 1965 over forty new patients have been treated. The marked increase in prevalence of this disease in males and also in females is evident countrywide and our experience has reflected this fact. This report is intended also as a reminder that lung cancer is a grim disease, presently curable only by surgery.

MATERIAL AND METHODS

One hundred and twenty-three of the 141 patients have been completely followed. The remaining eighteen or 12% have been lost to follow-up and are not hereinafter included. Our aim in treatment of these patients was strictly palliative. There was no expectation that we might significantly improve overall survival. A classification of three categories was set up. The first category includes those who were able to function normally. A second category includes those who were ambulatory but unable to work, and in a third category were placed all patients who were incapacitated. All of our patients were classified at the onset of radiation treatment. All of the patients who were treated had symptoms from their tumor. The most common symptom was pain, but persistent cough, hemoptysis, superior vena caval obstruction and dyspnea were other reasons for instituting treatment. It is important to note here that records were not well standardized and the information concerning each patient's subsequent course was often inferred as best we could from the available data. Some inadequacy of this type can be expected from any retrospective study, and especially here, where many of the patients were later followed by their own physicians in distant parts of the state.

The patients ranged in age from 31 to 82 and the average age at the date of diagnosis was 53.5 years. Nineteen or 15% of the patients were female. The histologic type of tumor present was not considered a criterion for radiation therapy. The majority of cases were of the undifferentiated or epidermoid variety, with an occasional adenocarcinoma. The predominance of males together with the large number of the former histologic types coincides with other reported series of bronchogenic carcinoma.[1,2,3]

As in other centers[4,5] symptomatic relief with radiation therapy is obtained in 65% to 85% of patients. At the Maine Medical Center, until 1963, treatment was exclusively with orthovoltage radiation, but since that time Cobalt-60 gamma teletherapy has been available and used preferentially. This is due to the greater ease with which a deep tumorstatic dose can be delivered, and the significant skin-sparing effect of supervoltage radiation. Orthovoltage radiation continues to be useful, particularly when osseous metastasis is present.

RESULTS

At the end of the ten-year period only eight of 123 patients remained alive. One was incapacitated and seven were apparently symptom-free. The longest survival was 73 months, the shortest was seven months. Since June of 1965, two of those eight patients have died, and they are not included in the following statistics.

The amount of radiation delivered to the mediastinum or lungs varied considerably. The usual dose was from 2000 to 4000 roentgens in air. The average dosage was 3500 roentgens per patient. Many patients were too debilitated to complete their treatment, while several were treated on more than one occasion. Thirty-three patients had extrathoracic radiation for symptoms of metastatic disease, averaging 3100 roentgens in air. Because of penetration factors tumor dose was approximately one-half the air dose in these patients.

Twenty-five of the patients accepted for treatment died within four weeks of the onset of radiation therapy. This represents 20% of the total number treated and includes many who were too ill to complete treatment. Many of these patients were so near death when treatment was begun that last-ditch efforts were fruitless. These patients are included in the statistics.

In the categories described, little evidence of upward movement could be demonstrated among our patients as a result of radiation therapy. The 32% of our patients who underwent therapy while "functioning normally" were in that category for an average of 2.6 months. These patients were later included in the next lower category of "ambulatory, but unable to work", where 72% of our patients remained for an average of 3.7 months. All of the patients are included in the lowest category where the average length of incapacitation was 2.3 months. These statistics are listed in Table 1. It is hoped that we can demonstrate upward mobility in these categories in the prospective study now underway.

As is widely known, definitive surgery offers the only

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TABLE 1

	Average Time
Working and well (37 patients)	2.6 months
Ambulatory, unable to work (83 patients)	3.7 months
Incapacitated, requiring nurse care (115 patients)	2.3 months
Length of survival from diagnosis (123 patients)	8.6 months

TABLE 2

Primary Therapy	*Length of Survival*				
	Less than 1 year	*More than 1 year*	*More than 2 years*	*More than 3 years*	*More than 5 years*
Definitive Surgery (Pre-palliative)					
22 patients	27%	36%	23%	14%	0
Radiation Alone (Palliative)					
36 patients	81%	11%	6%	2%	0
Radiation Treatment with known metastasis					
65 patients	85%	12%	0	0	2%

real hope of cure with present methods, but only if undertaken early while the tumor is asymptomatic. The results of therapy in our patients who had also undergone definitive surgery were somewhat better because they were operable and treated earlier in their course than those who had radiation only. Seventy-three percent of those who had prior surgery lived longer than one year, and a considerable number lived longer than two years. Their survival after the onset of radiation therapy was not different from the other nonsurgically treated patients. Few differences existed among those who had radiation only, with and without metastases at the time of treatment. These statistics are listed in Table 2.

The overall average length of survival from time of diagnosis in all patients whom we treated was 8.6 months. One patient is alive after five years but only 20% lived longer than one year from the time of diagnosis. Half of these patients were from the surgically treated group, and this reflects the fact that these patients had operable lesions at their original thoracotomy.

Discussion

Documentation of survival *quality* is admittedly difficult, especially in a retrospective study. Our records confirm this fact and we have now undertaken a prospective study to attempt more accurate evaluation of these subjective factors in our patients. Treatment now is almost exclusively with Cobalt-60 gamma teletherapy and in some cases dosage as high as 6000 rads is being delivered to the tumor. We are pursuing three objectives. As before, palliation is being attempted, and in addition, we have begun delivery of preoperative radiotherapy in selected cases. A few patients, considered inoperable, are being given high radiation dosage in an attempt to completely eradicate the tumor with radiation. Experience here and elsewhere[6] suggests this as an improvement over present practices in the surgically incurable patient.

An unexpected corollary that resulted from this study is the finding that survival statistics are also definitely affected by radiation treatment. Even though our avowed intent in all patients until now has been only palliation of symptoms, comparison with series of untreated lung cancers reveal that length of survival is also improved. Three studies of untreated patients, each describing results in over two hundred cases, reveal an average survival of 3.5 months from time of diagnosis.[5,7,8] In our patients, the average survival from time of diagnosis was 8.6 months, with a 5.2 month survival documented from the onset of radiation therapy. If, armed with this knowledge, we are able in the future to document improved *quality* of survival from radiation treatment, progress in the treatment of this almost invariably fatal tumor will have been made.

Conclusions

We have documented qualitative survival in lung cancer patients at the Maine Medical Center treated with palliative radiation therapy. The retrospective nature of the study prevented demonstration of upward mobility in our treatment categories. Many patients are referred very late in their course for last resort treatment and this fact tends to further prejudice a dismal outlook. A significant increase in length of survival has been documented in comparison to the untreated patient. A further prospective study is now underway, with some patients receiving treatment preoperatively and, in an occasional patient, treatment for cure with radiation is being attempted.

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Saphenous Vein Bypass Grafts in Femoro-Popliteal Occlusive Disease

FERRIS S. RAY, M.D.* AND C. PHILIP LAPE, M.D.**

Occlusive disease of the femoro-popliteal arteries continues to be one of the most common lesions encountered in the field of peripheral vascular surgery. In spite of increased experience and advances in surgical techniques, there still exists some disagreement in regard to the most reliable and successful method of correcting these lesions.

The credit for introducing the bypass principle in occlusive vascular disease goes to Kunlin[1] of Paris who in 1948 was the first one to introduce the use of the saphenous vein as a long bypass from the common femoral to the popliteal artery. Autogenous vein grafts were explored in this country by Julian[2] and others[3,4] in the early 1950's. Unfortunately they experienced a high failure rate when these techniques were used in occlusive disease. In 1957 Lord[5] reported only a 50% success rate with the use of the bypass vein graft. Although enthusiastic about the technique, his best results appeared to be in cases of trauma or in excision of aneurysms where only a short segment of diseased artery was involved.

Thromboendarterectomy has been extensively employed in occlusive disease of the femoro-popliteal vessels and still has many enthusiasts.[6,7,8] The accumulated experience of most surgeons, however, has indicated that this method is followed by a disappointingly high incidence of re-occlusion presumably due to fibrosis of the remaining vascular tissue.

The use of homologous human arteries was widespread for a time and several methods of banking human blood vessels were in common usage for many years. By 1959, however, surgeons everywhere were reporting late rupture, thrombosis and aneurysm formation in homografts which had initially been accepted and functioned ideally.

All of the foregoing experiences produced a tremendous interest and enthusiasm for the development of synthetic prostheses suitable for arterial replacement. When used in reconstruction of small blood vessels, however, the results using prosthetic grafts have been poor. Edwards[9] and others have reported a 50 to 60% late occlusion rate of synthetic grafts when used in the femoro-popliteal area.

Re-exploration of the capabilities of the autogenous vein graft had been urged by Linton for many years since he felt the original poor experience with this method was due to technical factors rather than to intrinsic limitations of the method itself.

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TABLE 1

CLINICAL MATERIAL

Total Cases	80
Femoro-femoral	3
Femoro-popliteal	77
Age (37 to 90) – Average Age 63	
Indications for Operation	
Claudication with or without Rest Pain	53
Ischemic Necrosis (Amputation Threatened)	27 (33%)

During the past five years, we have participated in what appears to be a general trend among vascular surgeons and have increasingly utilized the saphenous vein as a bypass graft in the reconstruction of occlusive lesions below the groin. The purpose of this paper is to present our experience with this operation.

CLINICAL MATERIAL

Since 1961, we have performed 80 saphenous vein bypass grafts for occlusive disease of the femoro-popliteal arteries. The indications for surgery were marked intermittent claudication, rest pain, or focal ischemic necrosis. Twenty-seven patients, over one-third of the total group, were threatened with a major amputation because of frank gangrene. The patients ranged from 37 to 90 years of age with an average age of 63. Clinically all patients had femoral pulses with no pulses distal to the femoral level. Femoral angiograms were performed on all cases to determine operability. The preoperative angiogram often revealed only the popliteal or one tibial artery to be patent. This was felt to represent a poor "runoff" and was frequently found in patients with frank gangrene or severe rest pain. At surgery we have often found a much better "runoff" than one might expect from the preoperative x-ray. We therefore agree with Linton,[10] Mannick,[11] and others that if even a short segment of distal popliteal artery or one of its major branches is displayed by angiography, one should not carry out amputation prior to popliteal exploration, since successful bypass grafting may still be possible.

COMPLICATIONS

The complications following operation are listed in Table 2. There were three deaths, all in elderly females. Two of the three patients were facing a thigh amputation because of gangrene associated with marked ischemic neuritis. In all three cases distal circulation was restored but death occurred on the 14th postoperative day in two instances; autopsy demonstrated a pulmonary embolus in one patient and an acute myocardial infarc-

TABLE 2

COMPLICATIONS

Deaths		3 (3.7%)
Female – Age 78 – Pulmonary Embolus	1	
Female – Age 75 – Myocardial Infarction	1	
Female – Age 80 – ? Pulmonary Embolus Sudden Death at Home 1 Month Post-op.	1	
Wound Complications		7
Sepsis (Major)	3	
Sepsis (Minor)	3	
Lymph Fistula	1	
Other		
Phlebitis	1	
Phlebitis with Pulmonary Embolus	1	

TABLE 3

RESULTS

(Follow-up – 1 Month to 5 Years – Avg. 2 Years)

Total Cases 80

	No.	Percentage
Success	69	86
Failures	11	14
Immediate (5)		
Delayed (6)		

TABLE 4

ANALYSIS OF FAILURES

Total Cases		11
Immediate Failure (5)		
Postoperative Death	2	
Sepsis	1	
Probable Poor Outflow	2	
Delayed Failure (6)		
Died at Home – 1 Month Post-op ? P.E.	1	
Stenosis of Distal Anastomosis	1	
Probable Progression of Distal and Proximal Disease	4	

tion in the other. The third death occurred suddenly at home one month postoperatively, perhaps as a result of pulmonary embolus.

There were three cases of major wound sepsis resulting in thrombosis of only one bypass graft. The two other cases of gross infection were treated successfully without loss of graft function and both have remained patent to date. Both cases were treated with radical drainage and debridement with specific antibiotics in high doses. Minor wound problems have diminished in the past few years as a result of better planning of the incisions and other refinements in technique.

RESULTS

The overall results are tabulated in Table 3. The long term success rate was 86% with 14% failures. The possible causes of failure are analyzed in Table 4. Two cases of immediate postoperative thrombosis were successfully reoperated and have continued to remain patent and function properly. These were felt to be secondary to technical factors, a small vein in one instance and partial occlusion at the popliteal bifurcation in the other. The cause of delayed failure was felt to be progression of distal or proximal disease in four cases and in one case stenosis of the distal anastomosis was demonstrated.

TABLE 5

RESULTS OF PATIENTS WITH GANGRENE

Total No. Patients with Gangrene	*Failures*	*Success*
27	6 (26%)	21 (74%)

Successful Cases (21)	Pathology	Result
11	Gangrenous Ulcers	Healed
1	Gangrenous Ulcer (Heel)	Healing without pain
3	Infected Gangrene (1 Toe)	Toes Amputated – Healed
1	Gangrenous Toes	Transmetatarsal Amputation – Healed
1	Grangrenous Toe	Stable – pain free
2	Infected Gangrene (Toes)	Open Transmetatarsal Amputation Healed
2	Extensive Gangrene	Below Knee Amputation

Failures (6)	Pathology	Result
1	Extensive Gangrene (Remaining Leg)	Death (Myocardial Infarction)
1	Gangrene (Big Toe)	Death (Pulmonary Embolus)
1	Gangrene (Foot)	Graft Worked 10 Months – Amputation
1	Gangrene (Toes)	Graft Worked 6 Months – Re-opened with Dacron Graft
1	Extension Gangrene age 90	Graft Worked 4 Months – Died in N.H. without amputation – no improvement
1	Gangrene (Heel)	No improvement – AK amputation

The results in patients with frank gangrene are listed in Table 5. Even in these cases there was a 74% success rate in spite of a poor outflow tract in most instances. In 11 cases the gangrenous ulcers completely healed. Five cases required local toe amputations and three transmetatarsal amputations were necessary all of which have healed. In two cases the gangrenous process of the foot was so extensive that healing was unlikely and a below knee amputation was required. It should be noted that even in cases of graft failure the patients were not made worse and amputations were not hastened.

DISCUSSION

We have been impressed, as have others,[10,11,12] that late occlusion of venous bypass grafts is rare if early postoperative function is established. In spite of progression of proximal and distal arteriosclerotic disease with very slow graft flow rates, Mannick and Jackson[12] have shown that the majority of vein grafts will remain patent and function successfully. Most early postoperative failures appear to be secondary to technical factors or a combination of inadequate inflow or outflow. Continued improvement in technique and careful selection of cases should further eliminate these early failures.

At exploration we have often found more disease in the femoral and popliteal vessels than was demonstrated on the preoperative angiogram. For this reason and because of the progressive nature of the arteriosclerotic disease in the superficial femoral artery, we now attempt to span the entire superficial femoral artery from the common femoral to the distal popliteal artery just above the bifurcation. To accomplish this, a long graft of fairly good caliber is required. Quite often the saphenous vein will bifurcate at the mid thigh and the distal portion will be less than 4 or 5 millimeters after distention. In this situation we have used the larger portion of the vein as a bypass graft combined with endarterectomy of the upper superficial femoral artery, utilizing the smaller segment of vein as an onlay patch graft. We have fortunately found a fairly satisfactory saphenous vein except on three occasions in which the above technique was employed. In instances where the saphenous vein has been completely or partially removed, the contralateral saphenous vein can be utilized.

The long term behavior of autogenous vein grafts appears to be quite satisfactory. Linton,[10] and others have performed postoperative angiograms as long as six years after implantation which show no dilatation or aneurysmal changes of the vein graft.

Summary

The long term patency rate of 86% has confirmed our impression that the autogenous saphenous vein bypass graft is the present operation of choice in occlusive disease of the femoral and popliteal arteries. In recent years Linton and others have reported long term success rates similar to ours with the use of the autogenous vein bypass.

Whenever possible, the entire superficial femoral artery should be bypassed from the common femoral to the distal popliteal artery.

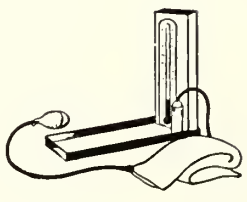
The success rate of 74% in patients with frank gangrene would appear to justify a continued aggressive approach to the leg that is threatened with an early major amputation.

We also feel that the results of this procedure today are good enough to justify recommending this operation not only to patients whose limb or livelihood is threatened but also to patients with aggravating claudication that interferes with activities which they wish to enjoy.

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MOLECULAR REMODELING—

laboratory exercise or clinical necessity?

More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors—in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9, 10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumethiazide a rather dramatic reduction in dose range is accomplished. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effective antihypertensive agent.

Contraindications: Severe renal impairment; previous hypersensitivity.

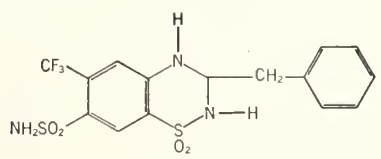
Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c̄ K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

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COMMISSIONER

State Of Maine

Department of Health and Welfare

State of Maine
Department of Health and Welfare
Division of Tuberculosis Control

Report on New Active Cases of Tuberculosis
1965

A. FORM AND EXTENT OF DISEASE AND BACTERIOLOGICAL STATUS

Form and Extent of Disease	*Number of Cases*	*BACTERIOLOGICAL STATUS* *Positive*	*Negative*	*Undetermined*
Total	142	85	31	26
Pulmonary Total	119	77	27	15
Primary	12	4	2	6
Minimal	35	13	20	2
Moderately Advanced	40	34	4	2
Far Advanced	27	23	1	3
Extent Not Specified	5	3	0	2
Unexplained Pleurisy	5	0	2	3
*Other Nonpulmonary	18	8	2	8
Form Not Specified				

B. SOURCE OF NEW CASE REPORTS

	142	*Number of Cases*
Total		
Private Physicians		77
Tuberculosis Sanatoria (Public & Private)		2
General Hospitals		20
Chest Clinics		11
Mental Institutions		28
Death Certificates		3
Transferred from Other States		1
Other		

C. AGE, RACE AND SEX

	NUMBER OF CASES					
		White		*Nonwhite*		*Race or Sex*
Age	*Total*	*Male*	*Female*	*Male*	*Female*	*Not Specified*
Total (Same as in A)	142	89	49	3	1	
Under 5 Years	3	2	1			
5-14 Years	16	11	5			
15-24 Years	21	10	11			
25-44 Years	38	21	15	1	1	
45-64 Years	43	29	13	1		
65 and over	21	16	4	1		
Not Specified						

D. NUMBER OF PATIENTS ADMITTED TO SANATORIA	*Total* 127	*V.A.* 7	*State* 120
Number of these who are re-admissions	39	5	34
Number of patients discharged from sanatoria	126	11	115
Number of these who left against advice	22	2	20
Number discharged AA with positive sputum	8	0	8
% of patients leaving against advice	17.4%	18.1%	17.3%

*E. NONPULMONARY TYPES

Type	*Number*
TB Glands	4
TB Salpingitis	1
Endobronchial TB	1
Miliary TB	4
G.U. TB	3
TB Bones and Joints	4
TB Peritonitis	1

Total Tuberculosis Cases in Each County, Hospitalized and at Home By Activity Status As Of 12/31/65

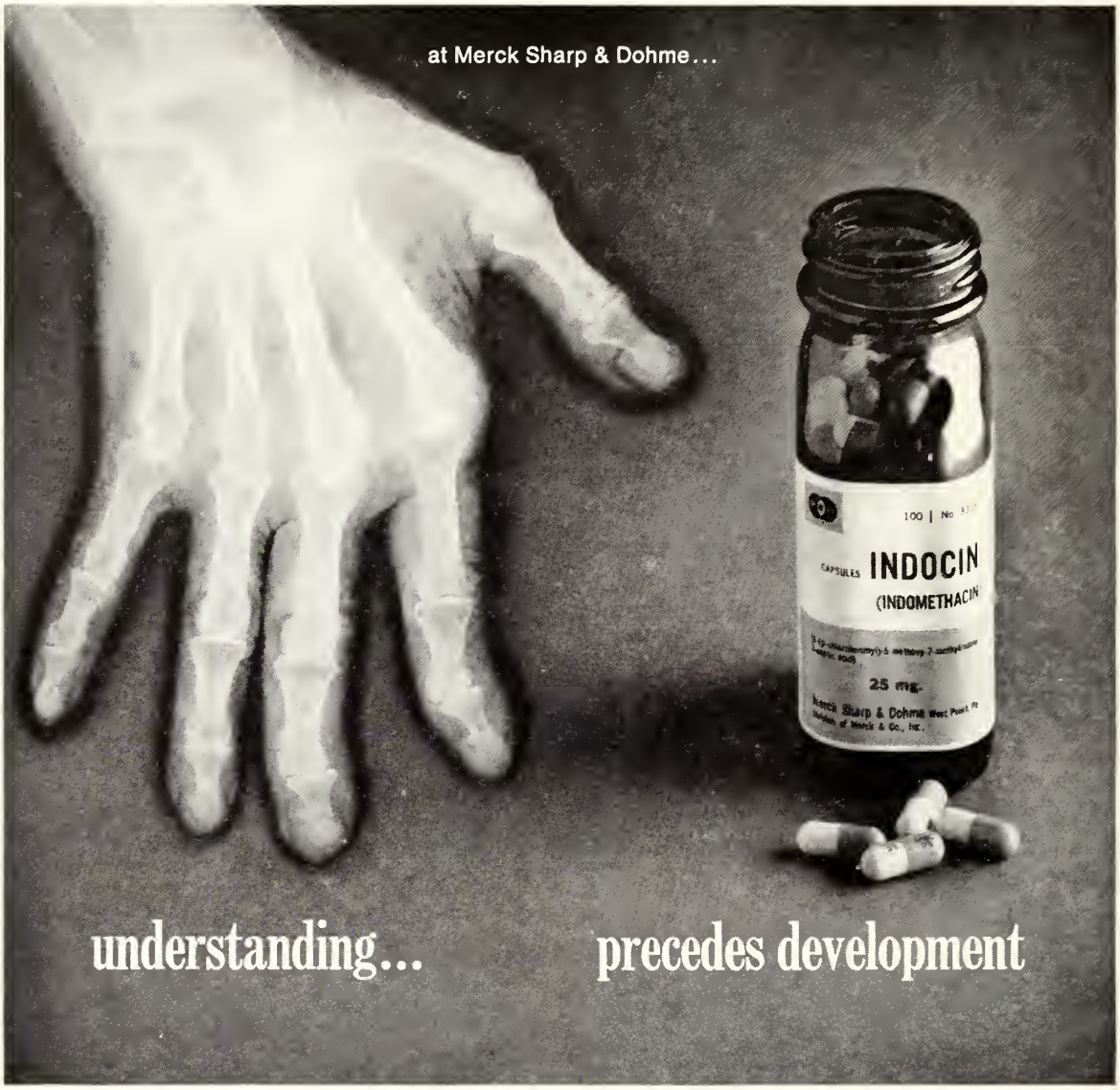
					NON-HOSPITALIZED		
COUNTY	*Totals*	*Rate*	*Hospitalized*	*Active*	*Inactive*	*Undetermined*	*Deceased*
Androscoggin	10	11.0	3	2	4	1	0
Aroostook	30	27.9	2	21	4		3
Cumberland	13	6.8	2	7	2		2
Franklin	2	9.4			2		
Hancock	3	9.2		2	1		
Kennebec	8	8.7	1	5	1		1
Knox	8	27.3		5	3		
Lincoln	3	15.9		2			1
Oxford	4	9.1	2	2			
Penobscot	16	12.6	10	3	1		2
Piscataquis	0	-					
Sagadahoc	2	8.7	1				1
Somerset	4	9.9	1		2		1
Waldo	2	8.7	1	1			
Washington	3	9.3		3			
York	6	5.8	1	4	1		

Tuberculin Testing (All Types) Entering – Seventh Grade and Others School Year 1965-1966

State of Maine

	Total Enrollment	*Total Tested*	*Percent Participation*	*Number Reactors*	*Percent Reactors*	*Reactor Rate*
Entering Grades	20,451	17,290	84.5%	53	0.306%	3.1
Grade 7	15,992	13,589	84.9%	132	0.971%	9.7
Others*	3,583	2,542	79.4%	36	1.416%	14.2

*Other consists of elementary grades, high school, special group (retarded) and school personnel.



The synthesis of cortisone was accomplished by Merck Sharp & Dohme in 1948—the famous "Compound E" used by Dr. Philip Hench in his historic experiment at the Mayo Clinic.

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Division of Merck & Co., INC., West Point, Pa.

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Indications: Chronic and acute rheumatoid arthritis, rheumatoid (ankylosing) spondylitis, degenerative joint disease (osteoarthritis) of the hip, and gout.
Contraindications: Active peptic ulcer, gastritis, regional enteritis, or ulcerative colitis. Safety in pregnancy has not been established. Not recommended for pediatric age groups.
Warning: Patients who experience dizziness, lightheadedness, or feelings of detachment on INDOCIN should be cautioned against operating motor vehicles, machinery, climbing ladders, etc. Use cautiously in patients with psychiatric disturbances, epilepsy, or parkinsonism.
Precautions and Adverse Reactions: Most commonly, headache, dizziness, lightheadedness, G.I. disturbances. The C.N.S. effects are often transient and frequently disappear with continued treatment or reduced dosage. The severity of these effects may occasionally require cessation of therapy. G.I. effects may be minimized by giving the drug with food or with antacids or immediately after meals. Ulceration of the stomach, duodenum, or small intestine has been reported and, in a few instances, severe bleeding with perforation and death. Gastrointestinal bleeding with no obvious ulcer formation has also been noted; INDOCIN should be discontinued if G.I. bleeding occurs. As a result of G.I. bleeding, some patients may manifest anemia, and for this reason periodic hemoglobin determinations are recommended. Rare reports of effects not definitely known to be attributable to INDOCIN include bleeding from the sigmoid colon (either from a diverticulum or without a known previous pathologic condition), perforation of preexisting sigmoid lesions (diverticulum, carcinoma), and hematuria. In other rare cases, a diagnosis of gastritis has been made while the drug was being given. One patient developed ulcerative colitis, and another, regional ileitis, while receiving INDOCIN; when the drug was given to patients with preexisting ulcerative colitis, there was an increase in abdominal pain. Infrequently observed side effects may include drowsiness, tinnitus, mental confusion, depression and other psychic disturbances, blurred vision, stomatitis, pruritus, edema, and hypersensitivity reactions. Slight BUN elevation, usually transient, has been seen in some patients, although the preponderance of evidence indicates that INDOCIN does not adversely affect renal function, even in patients with preexisting renal disease. Nevertheless, renal function should be checked periodically in patients on long-term therapy. Leukopenia has been seen in a few patients. Transient elevations in alkaline phosphatase, cephalin-cholesterol flocculation, and thymol turbidity tests have been observed in some patients and, rarely, elevations of SGOT values; the relationship of these changes to the drug, if any, has not been established. As with any new drug, patients should be followed carefully to detect unusual manifestations of drug sensitivity.
Before prescribing or administering, read product circular with package or available on request.

Maine Medical Association

SPECIAL COMMITTEES 1966-1967

Special Committees for 1966-1967 as appointed by the President, George E. Sullivan, M.D. of Fairfield

(Continued from August issue)

Committee on Disaster Medical Care

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4th — Allan J. Stinchfield, M.D., P.O. Box 343, Augusta
5th — Joseph H. LaCasce, M.D., 50 Union St., Ellsworth
6th — William M. Blackwell, M.D., Millinocket Community Hospital, Millinocket
Members-at-Large
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Clark F. Miller, M.D., 46 Madison St., Auburn
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Nelson P. Blackburn, M.D., Bath Memorial Hospital, Bath
Robert D. Wakefield, M.D., St. Mary's General Hospital, Lewiston
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Hays G. Bowne, M.D., 9A Main St., Farmington
Harry M. K. Peddie, M.D., Doctors Park, 89 Hospital St., Augusta

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Ella Langer, M.D., 2 Chapel St., Augusta
Benjamin L. Shapero, M.D., 431 State St., Bangor
Maurice Ross, M.D., 372 Main St., Saco

School Health Committee

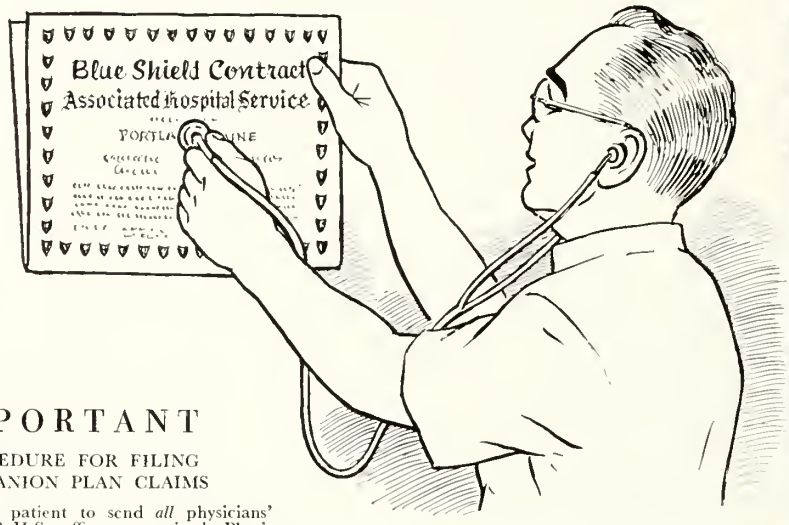
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Sidney R. Branson, M.D., 37 Main St., South Windham
Lloyd G. Davies, M.D., 78 Main St., Fryeburg
James A. MacDougall, M.D., 303 Penobscot St., Rumford

Committee on Aging

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Richard T. Chamberlin, M.D., 14 Gilman St., Waterville
Niles L. Perkins, M.D., Box 1489, Portland

Continued on Page 220

Blue Shield Plan
OF THE
Associated Hospital Service of Maine



IMPORTANT

PROCEDURE FOR FILING
COMPANION PLAN CLAIMS

Instruct your patient to send *all* physicians' bills to the A.H.S. office as received. Physician services must be itemized and their "reasonable charges" for each item indicated on the bills. A brief description of the injury or illness treated should also be shown. *The Companion Plan contract number and the Social Security or Government Identification number must be entered on the bill by the patient or the doctor.* For prompt direct payments of the Companion Plan benefits the bill should be issued to the patient in duplicate, one for Blue Cross-Blue Shield use and one for the Part B intermediary if you are not making claim on the Government Form SSA 1490.

If the bill is not receipted, payment will be made directly to the doctor on the regular semi-monthly Blue Shield check. The notation "C.P." for Companion Plan will be shown under the heading "Coverage" on the check voucher. This procedure will assure the doctor of the most prompt payment.

If the bill is receipted, payment of benefits will be made to the patient.

Medicare "Explanation of Benefits Form" may be used by the patient making claim for benefits but should be accompanied by itemized physicians' bills for all services incurred during the calendar year.

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Maine Heart Association Notes
116 State Street, Augusta, Maine

The Second Heart Sound in Pulmonary Embolism and Pulmonary Hypertension

. . . In acute massive embolism and in severe chronic blockage of the main pulmonary arteries, the second heart sound is usually very widely, and often fixedly, split, in the absence of any other detectable cause. When borderline, the abnormal splitting can usually be increased and fixed by mild exercise. The "second heart sound sign" has been helpful in finding cases of massive thromboembolism previously missed, in evaluating the course of patients treated medically, and, recently, in deciding how urgently pulmonary embolectomy is needed. . . . In 18 proved cases of massive acute pulmonary embolism or chronic pulmonary artery thrombosis with pulmonary hypertension, the second heart sound was abnormally split in expiration; . . .

In chronic obstruction of the pulmonary arteries, the hemodynamic factors most readily correlated with the degree of splitting of the second heart sound were the height of the pulmonary arterial systolic pressure and the presence of an elevated right ventricular diastolic pressure at rest.

By contrast, extreme splitting of the second sound is rarely encountered in other causes of pulmonary hypertension and right ventricular failure. This is notably true of rheumatic heart disease. Primary pulmonary hypertension and small repeated peripheral pulmonary emboli also often do not produce comparable splitting in spite of gross accentuation of the pulmonic component.

Whatever its mechanisms, the clinical usefulness of the second heart sound sign has been established in a variety of circumstances. . . . Absence of the sign should make one hesitate to advise pulmonary embolectomy. Yet we do not mean to imply that the second sound is abnormally split in all cases of significant pulmonary embolism. Emboli may be quite important, and in some circumstances even life-threatening, without being massive. . . . The presence of bundle branch block or any other cause of wide splitting obviously vitiates the sign.

Submitted by Jacob B. Dana, M.D.
Reference—B. W. Cobbs, Jr., M.D. et al, American Heart Journal, Vol. 71, pages 843-844, 1966



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Indications: Meprobamate is effective in relief of anxiety and tension states. Also as adjunctive therapy when anxiety may be a causative or otherwise disturbing factor. Although not a hypnotic, meprobamate fosters normal sleep through both its anti-anxiety and muscle-relaxant properties.

Contraindications: Previous allergic or idiosyncratic reactions to meprobamate or meprobamate-containing drugs.

Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

Supplied: 'Miltown' (meprobamate) is available in two strengths: 400 mg. scored tablets and 200 mg. coated tablets. 'Meprotabs' (meprobamate) is available as 400 mg. white, coated, unmarked tablets. *Before prescribing, consult package circular.*

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AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Soft Tissue Grafts — Success or Failure

A scientist at the Scripps Clinic and Research Foundation in LaJolla, California is unraveling the secrets of cell compatibility and incompatibility.

The animal studies are helping to show why some tissue grafts are successful and how others fail. The research may be of value in transplanting human organs, and, eventually, in understanding how cancer survives in a hostile host.

This was reported recently by the American Cancer Society, which supports the research by Dr. Richard W. Dutton, an experimental pathologist.

When skin or some other tissue is transplanted to another animal, it starts to heal into its new position and function as if it were the animal's own. Quite soon, however, the animal's own tissues start to attack it and finally destroy and reject the graft. This response, the "homograft response", is an immunological reaction and is carried out by certain specialized cells of the body. These are the lymphoid cells, which are found in the spleen, lymph nodes and other organs of the lymphatic system.

This lymphatic system has a remarkable ability to distinguish friend from foe. One's own, or friendly, cells are not subject to lymph cell attack; disease-causing organisms and foreign cells, under ordinary conditions, are quickly done to death.

One of the most pressing problems in medical science is ignorance of how lymph cells distinguish friend from foe. When this secret is known, it may be possible to replace damaged or worn-out organs with transplants, to control a large number of diseases in which healthy structures come under antibody attack, and to treat cancers more effectively.

The lymphoid cells will also attack foreign cells when they are cultivated outside the body, and Dr. Dutton has made use of this fact to investigate some of these problems.

He has shown that the first thing that happens when the cells are placed in contact with the foreign tissue is that a small number of them start to multiply very rapidly. The size of this response depends on how "foreign" the foreign tissues are. The responses against the foreign substances or "antigens" of very distantly related cells are large, while there is no response at all against cells from identical twins.

By using special pairs of strains of mice that differ by a single gene that controls a transplantation antigen, it was shown that the test tube response is triggered by the same antigens that trigger skin graft rejection.

Studies are aimed at discovering which antigens are important in graft rejection, how the lymphoid cells "detect" foreign tissue and how they make their response.

Knowledge gained from a successful outcome of these studies may help to design ways of suppressing the response so that tissue grafting can be achieved or increasing the response in the hope the body may be able to destroy malignant tumors in the same way as they reject grafts.

American Cancer Society News Service Release

Necrologies

Gerald C. Leary, M.D.

1915-1966

Gerald C. Leary, M.D., 50, of Portland, Maine died on June 25, 1966. He was born in Lowell, Massachusetts on April 21, 1915, son of Nora H. and the late Jeremiah F. Leary.

Dr. Leary attended Lowell schools, graduated from Keith Academy, Holy Cross College in 1937 and received his medical degree from Tufts University School of Medicine in 1941. He interned at the St. John's Hospital in Lowell from 1941 to 1942 and then served in the U.S. Navy as Lieutenant Commander in the European and Pacific Theatres. He served his residencies at the New England Deaconess Hospital and the Pondville Hospital in Massachusetts.

Dr. Leary worked under the famous Dr. Papanicolaou at Cornell University in 1948 and then moved to Johnstown, Pennsylvania where he practiced medicine at the Conemaugh Valley Hospital from 1949 to 1951. In 1951 he came to Portland and served as chief pathologist at Mercy Hospital until his death.

He was a member of the Cumberland County Medical Society, the Maine Medical Association, the American Medical Association and the American Association of Pathologists. Dr. Leary was also the secretary of the Maine Society of Pathologists, head of the pathology department at the Knox County General Hospital and had been instructor in parasitology at Harvard Medical School.

Surviving besides his mother are one daughter, Miss Maureen B. Leary; three sons, Gerald C., Jr., Charles L. and Brian J. Leary, all of Portland; one brother, William Leary of Lowell and several nieces, nephews and cousins.

A Tribute to Dr. Leary*

Dr. Gerald Leary came to Portland in 1951 to serve as the first full-time pathologist at Mercy Hospital. He immediately began organization of the clinical laboratory and blood bank. An autopsy program to include Medico-Legal consultation at the Mercy Hospital and for York County was set up. His advice and counsel in this area was of valuable assistance to law enforcement agencies. Shortly after arriving in Maine, he was elected to membership in the Maine Pathology Society. His warm and congenial personality endeared him to all of us and because of his desire to be of assistance, he was elected Secretary in which capacity he served until illness in the Fall of 1965 which necessitated relinquishing this office. During his tenure in office he served the Society faithfully, intelligently, and accurately especially at a time when we were burdened with many decisions regarding the formulation of plans for the Medicare Program.

The membership of this Society is saddened by the untimely death of Dr. Leary. His generous and able contributions of advice and counsel at our meetings will be missed.

Joseph E. Porter, M.D.

*Presented at a meeting of the Maine Society of Pathologists, August 9, 1966.

David V. Mann, M.D.

1913-1966

David V. Mann, M.D., 52, of Camden, Maine died on June 3, 1966. He was born in Randolph, Massachusetts on July 19, 1913, son of Isaac R. and Elizabeth M. Mann.

Dr. Mann attended Milton High and Thayer Academy in Massachusetts, Acadia University, the University of Virginia and received his medical degree from the University of Virginia School of Medicine in 1942. He interned at the Central Maine General Hospital in Lewiston in 1942 and the Quincy City Hospital in Massachusetts from 1942 to 1943. Dr. Mann served in the Army Medical Corps with the Fifth Air Force in the South Pacific and in the occupation of Japan from 1943 to 1946.

He was located in Camden from 1946 to 1951 when he went to Harvard Medical School to complete graduate work in dermatology, acting as a fellow at the Massachusetts General Hospital. He then returned to Camden and practiced dermatology in Rockland, Bath and Augusta.

Dr. Mann was a member of the Knox County Medical Society, the Maine Medical Association, and the American Medical Association. He was also medical examiner of Knox County, a staff member of Knox County General Hospital, Camden Community Hospital, Bath Memorial Hospital, Augusta General Hospital, Waldo County General Hospital and Miles Memorial Hospital. He served as the Maine State Prison physician, the Camden school physician and town health officer of Camden and Rockport.

Dr. Mann had a wide scope of interests aside from the medical profession, including membership in the Maine Obsolete Automobile League, the Camden Historical Society and the National Association of Watch and Clock Collectors. He was also a lieutenant commander in the Wawenock Division of the United States Power Squadron.

Surviving are his widow, Mrs. Sarah Elizabeth Cunningham Mann; his mother of Camden; two sons, Carter Roel, a sophomore at Acadia University, Wolfeville, N.S. and Eric Wallace, a freshman at Camden-Rockport High School.

Letter to the Editor

To the Editor:

I am writing you regarding the impartial medical examination procedure which came into effect last November 30, when many changes were made in the Workmen's Compensation Act.

I was asked by Chairman Towle to communicate with you and to express the appreciation of the entire Commission to the members of the medical profession in Maine for their very prompt attention to our requests for scheduled examinations, the promptness of their reports, and the fact that the fees have been within a very reasonable structure without having to set up a fee schedule of any type. The impartial medical examination aspect is proceeding very well indeed and as can be readily understood, much of the credit belongs to the expeditious way in which the members of the medical profession are carrying out their part of the program.

It is all too often that a person or a group is not given a word of thanks or an expression of appreciation for what they are doing and we believe that such an expression is certainly in order in this instance, and we would very much appreciate your disseminating this information among your colleagues.

In the event we can be of any assistance to the medical profession, we sincerely hope they will make immediate contact with us and we take this opportunity to express our sincere appreciation for their splendid cooperation.

KENDALL H. DUNBAR
Rehabilitation Counselor
State of Maine Industrial Accident Commission
Augusta, Maine

News and Notes

Boston Medical Reports

The Bingham Associates Fund and the Postgraduate Medical Institute invite all physicians to participate in the educational television series for practicing physicians, BOSTON MEDICAL REPORTS — starting next month on educational television stations throughout the State of Maine. The series, designed to meet the needs and demands of Maine physicians, will bring the resources of university teaching hospitals and medical centers throughout the country to the physician in his own community and in his own hospital.

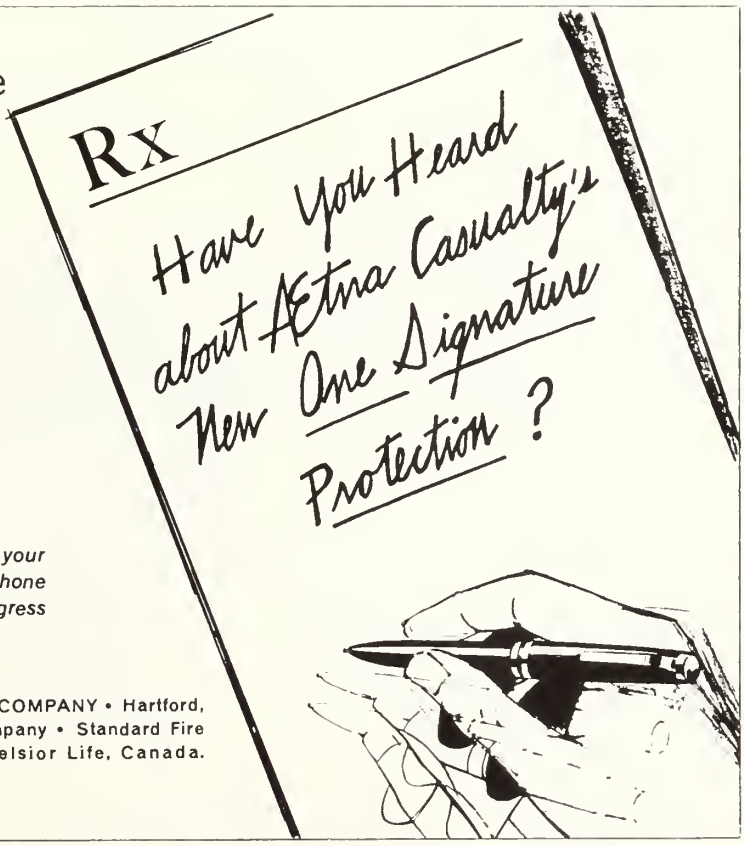
BOSTON MEDICAL REPORTS will begin its 1966-1967 season of 26 weekly programs on Tuesday, October 25, 1966 over WCBB, Channel 10, Lewiston; WMEB-TV, Channel 12, Orono; WMEM, Channel 10, Presque Isle; and WMED, Channel 13, Calais. In response to requests for more frequent telecasts each program will be shown three times each week: Tuesday at 12 noon, Wednesday at 10:00 p.m., and Thursday at 6:00 p.m.

Physicians residing in York County may view the series starting one week later, Tuesday, November 1, 1966, on

To members of the Maine Medical Association . . .

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WENH-TV, Channel 11, Durham, New Hampshire. Programs will be broadcast Tuesdays at 12 noon and Thursday evenings at 11:00 p.m.

Advance information including subjects to be covered, names and titles of participants, and abstracts and references will be sent out to all Maine physicians. For further information regarding BOSTON MEDICAL REPORTS please write Bingham Associates Fund, 171 Harrison Avenue, Boston, Massachusetts 02111.

M.M.A. SPECIAL COMMITTEES 1966-1967 — *Continued from Page 213*

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Paul A. Fichtner, M.D., 781 High St., Bath
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*Appointed by the Council of the M.M.A. on 8/7/66

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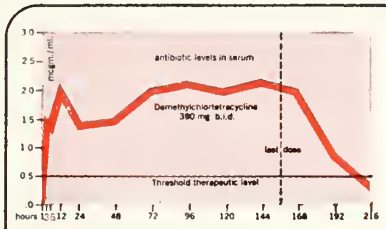
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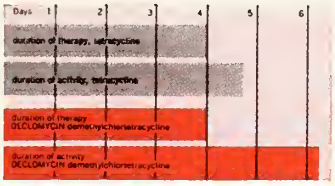
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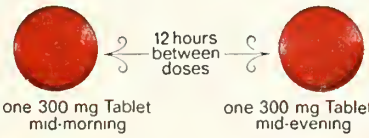


high activity

From Sweeney, W. M., Dornbush, A. C., and Hardy, S. M., Amer. J. Med. Sci. 243:296 (Mar.) 1962



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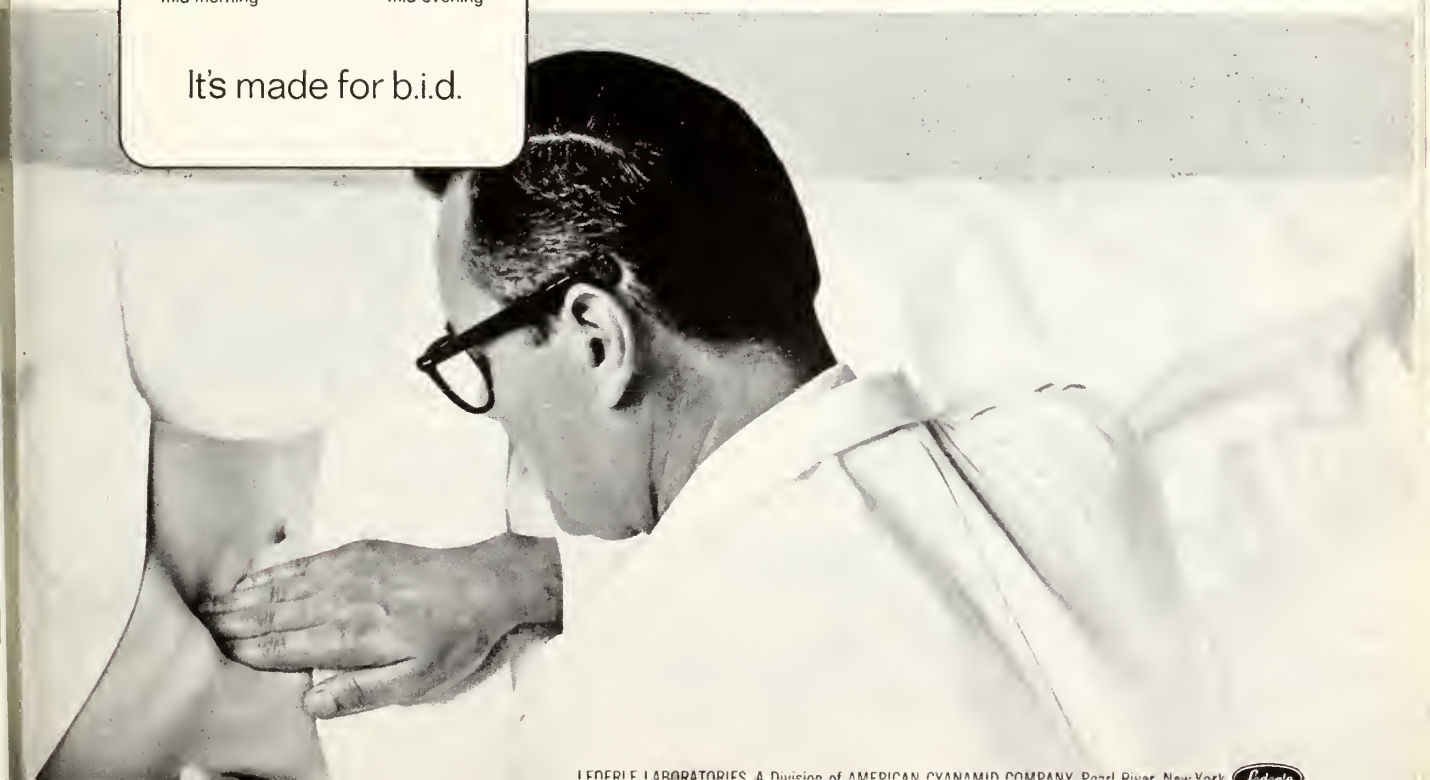
Contraindication—History of hypersensitivity to demethylchlortetracycline.

Warning—In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort.

Precautions and Side Effects—Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.



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Side effects have been minor. Occasionally gastric distress, weakness, sedation or dizziness occur. Reversible cholestatic jaundice has been reported on rare occasions. However, in 4,653 patients treated with chlormezanone, 97.7% had *no* side effects.[1] *Contraindication:* just one: sensitivity to aspirin. *Dosage:* Adults, usually 2 tablets three or four times daily. Children (from 5 to 12 years), 1 tablet three or four times daily.

1. Collective studies, Department of Medical Research, Winthrop Laboratories.

Winthrop WINTHROP LABORATORIES, NEW YORK, N. Y. 10016

summer 1966

DORSEY Season

A journal within a journal published quarterly in the interests of better medicine by Dorsey Laboratories, a division of The Wander Company, Lincoln, Nebraska. Address communications to Raymond C. Pogge, M. D., Director of Medicine.



this issue: emergency anesthesia and the common cold

When emergency anesthesia is complicated by the common cold

Barry Belonsky, M.D., F.A.C.A.
Staff Anesthesiologist, Hospital of The Albert Einstein College of Medicine, New York City



Medical facilities are often presented with unfamiliar patients who have unknown health histories. This is particularly true in emergency situations that arise due to accidents or acute illnesses. These cases may need prompt care requiring anesthesia, and if they involve colds, nasal allergies or other upper respiratory infections, can account for many complications which make up a major hazard during emergency anesthesia.

Administration of general anesthesia to a patient with a cold or upper respiratory infection is a hazardous undertaking. It should be avoided if at all possible. Indeed, the presence of U.R.I. is good reason for postponement of elective surgery.[1] In emergency surgery, regional or local block should be considered, but if general anesthesia is mandatory, it should be approached with utmost caution.

Since the attitude of "emergency surgery — hurry" has been replaced by "emergency surgery — watch out",[2] a knowledge of the complications is a great help in preventing them. Here is a brief outline of the problems involved and their treatment. Prevention of the complications is discussed later.

Complications during the induction of anesthesia

Most of the complications are a direct result of secretions and some a result of accompanying secondary infection. For example, *airway obstruction* due to excessive secretions occurs very commonly and is the direct effect of the cold. Respiratory exchange may be obstructed at any time during anesthesia because of excessive secretions, but is most likely to occur during induction. Suction apparatus must be available to overcome this.[3]

Excess secretions which stimulate and irritate the epiglottis and vocal chords can cause *laryngeal stridor and obstruction*. This can lead to complete laryngeal closure with resultant anoxia and death.

Bronchospasm and laryngospasm can result from secretions penetrating the bronchi and bronchioles. In laryngospasm, there are both inspiratory and expiratory stridor and difficulty in inflating the chest; in bronchospasm there is an expiratory wheeze, but not as much difficulty in inflation, although some resistance may be felt. Stridor is due to partial or complete closure of the vocal cords in spasm and the "crowing" sound is almost pathognomonic.

Secretions obstruct the nasal airways. This produces *difficulty in ventilation* through the mouth until the patient is deep enough to place an oral airway. An intravenous agent can be given to facilitate the induction of anesthesia.

Difficulties can arise if intubation is performed to ventilate the patient. For example, teeth can be broken by too vigorous attempts at intubation, or the intubation itself may be technically difficult due to secretions obstructing the view of the glottis. The postoperative sequelae of intubation ranges from mild laryngitis to pneumonia with atelectasis, and are seen far more commonly in patients suffering from colds than in normal patients.



Successive stages of laryngospasm which produce the characteristic stridor or "crowing" sound.



Progression of bronchioles into bronchospasm.

Complications during the maintenance of anesthesia *Bronchospasm* can occur in an unintubated patient due to secretions entering the bronchial tree from above, and acting as an irritant to the bronchi and bronchioles. Secretions accumulate quickly and the patient has to be suctioned continually. The whole cycle of coughing, bucking, laryngospasm and bronchospasm may ensue. The difficult decision here is whether it is better to suction the patient continually or to use an endotracheal tube which protects the cords and bronchi but introduces the risk of attendant complications.

Postoperative complications Postoperatively, complications can be more serious than even the intra-anesthesia complications, and occur much more frequently in a patient who has been intubated.[4]

Sore throat and pharyngitis can result both from the preoperative upper respiratory infection and from the drying of the mucous membranes which occurs during anesthesia.

Tracheitis and bronchitis often result from secretions trickling down the tracheobronchial tree.

Laryngitis is frequently seen in patients with upper respiratory infections who have been intubated. There is a significant increase in the incidence of laryngitis compared to that in patients without upper respiratory infections.

Subglottic edema is a condition which occurs mainly in children who have been intubated. This pathology results from an exudate developing in the areolar tissue just below the cords. Because of the small size of the child's trachea, even a 1 mm increase in size of the mucous membrane can severely impair the air passage. Children exhibit this by severe expiratory stridor and may even become cyanotic. This may so severely embarrass the child's breathing that it must be treated vigorously. Most authorities agree on the treatment[5,6,7,8] consisting of a high oxygen concentration in the inspired air (60%), plus high humidity (close to 100%). Adequate parenteral fluid intake and slight cooling of the body temperature (by a cooled oxygen tent) also help in mild cases. In severe cases, there may be hypoxia which increases the restlessness and the oxygen demand rises. Sedation is often necessary, although concomitant depression of the respiratory center is undesirable. An antihistaminic accomplishes this purpose well, and adds sedation. Since there is always a possibility that an allergic response plays a role in edema, some relief of the respiratory distress may occur. Steroids should be used to control inflammatory and allergic phenomena and swelling. If all this fails, and the patient is still restless and hypoxic, a tracheostomy should be performed immediately.

(concluded on following page)



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Cross section of trachea showing subglottic edema and lumen reduction due to mucous membrane congestion.

Pneumonia may also follow anesthesia administered to a patient with a cold. This can be caused by accumulated secretions becoming secondarily infected and causing consolidation of the lung. *Atelectasis* of the lung can result if one of the bronchioles becomes plugged by secretions, preventing aeration of the distal part of that lung. This is seen more frequently following upper respiratory infection because dry anesthetic gases aggravate the infection, causing secretions to change from watery to thick and viscid, and consequently difficult to suction.

Prevention of complications

The first rule to prevent complications, of course, is to use a regional or local anesthesia whenever possible. But when emergency surgery is a must, in spite of the presence of a cold, allergy, or upper respiratory infection, here are some ways to prevent complications.

Give nose drops preoperatively. This can help shrink the congested nasal mucous membranes and reduce secretions for better air passage. (Results of this method are sometimes unsatisfactory because of the short duration of effect or rebound congestion.) For longer effect, oral antihistamines with nasal decongestants are often given to provide and maintain a drying effect on secretions.

To clear the tracheobronchial tree, instruct the patient to cough preoperatively. Cold steam or water nebulizers effectively humidify the nasal, pharyngeal and bronchial passages and often make the patient more comfortable. Tenacious secretions become more watery under humidification, clear more thoroughly preoperatively and are more easily suctioned from the airway during anesthesia.

Give intravenous fluids to those patients who appear dehydrated due to a cold. In a well hydrated patient the respiratory tract secretions are less viscid and more watery. This is particularly true in asthmatics.

Summary:

Administration of emergency anesthesia to a patient with a cold or upper respiratory infection can lead to a chain of events that may result in increased postoperative morbidity and even death. This is because of the excess secretions formed in these conditions. Preoperative measures to prevent or reduce these secretions should be undertaken and will result in smoother and safer anesthesia.

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The means of therapy

The value of hormone therapy for this indication is of course well established. Both androgen and estrogen are known to inhibit the production and secretion of the lactogenic hormone by the anterior pituitary. As estrogen levels decline sharply at parturition, lactogenesis is established. When androgen and estrogen are administered to the patient before the release of the lactogenic hormone lactation and breast engorgement are usually prevented.

The time of therapy

The time of administration of this combined medication is crucial; it must be given early enough to suppress the pituitary prolactin and last long enough to permit physiologic readjustment during the puerperium. Excellent results are most often seen when therapy is administered before the onset of the second stage of labor.

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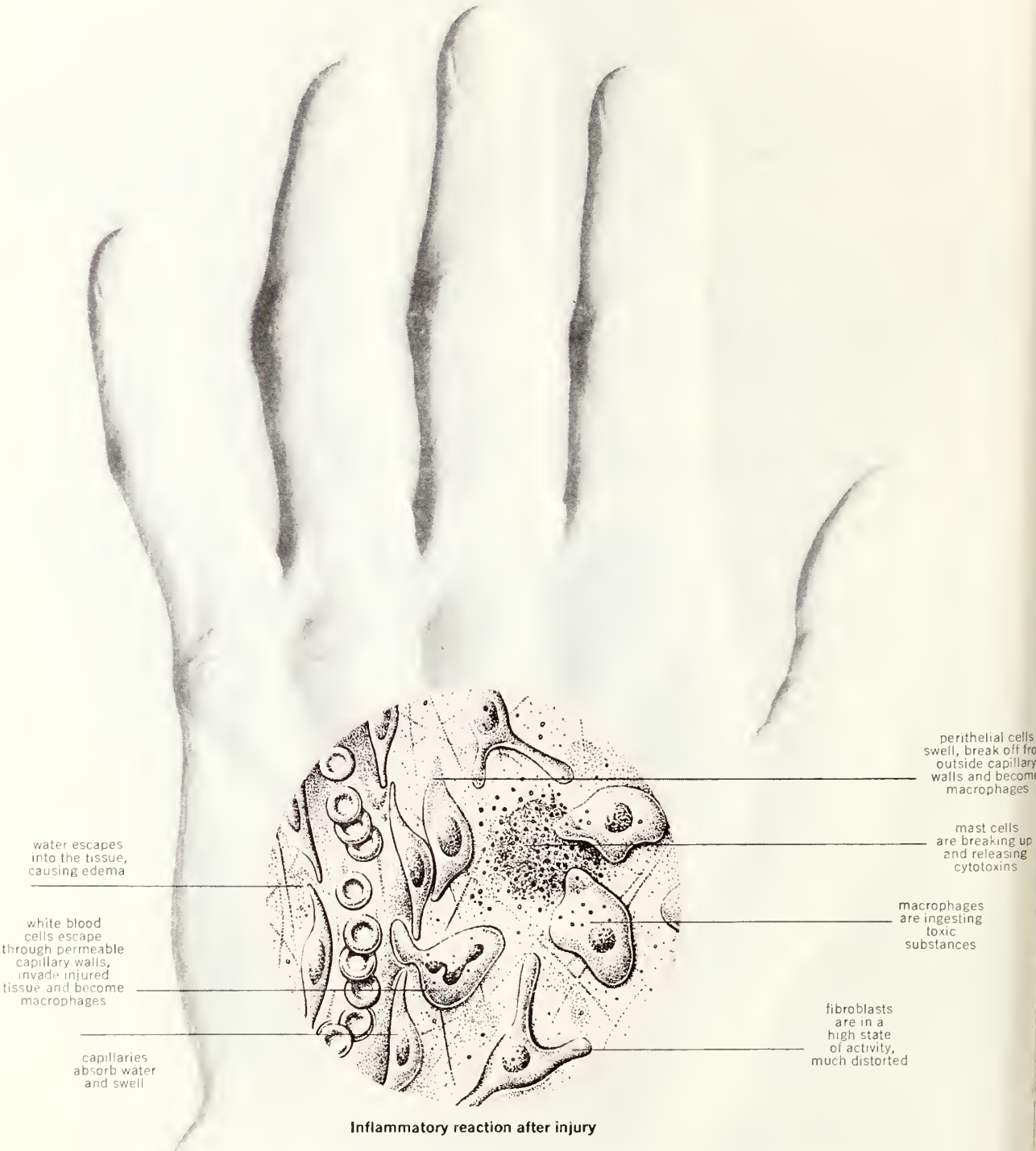
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References 1. Kanee, B.: Canad Med Ass J 88:999 (May 18) 1963. 2. Scholtz, J. R.: Calif Med 95:224 (Oct.) 1961. 3. Jansen, G. T., Dillaha, C. J., and Honeycutt, W. M.: Arch Derm 92:283 (Sept.) 1965.

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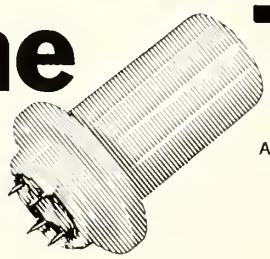
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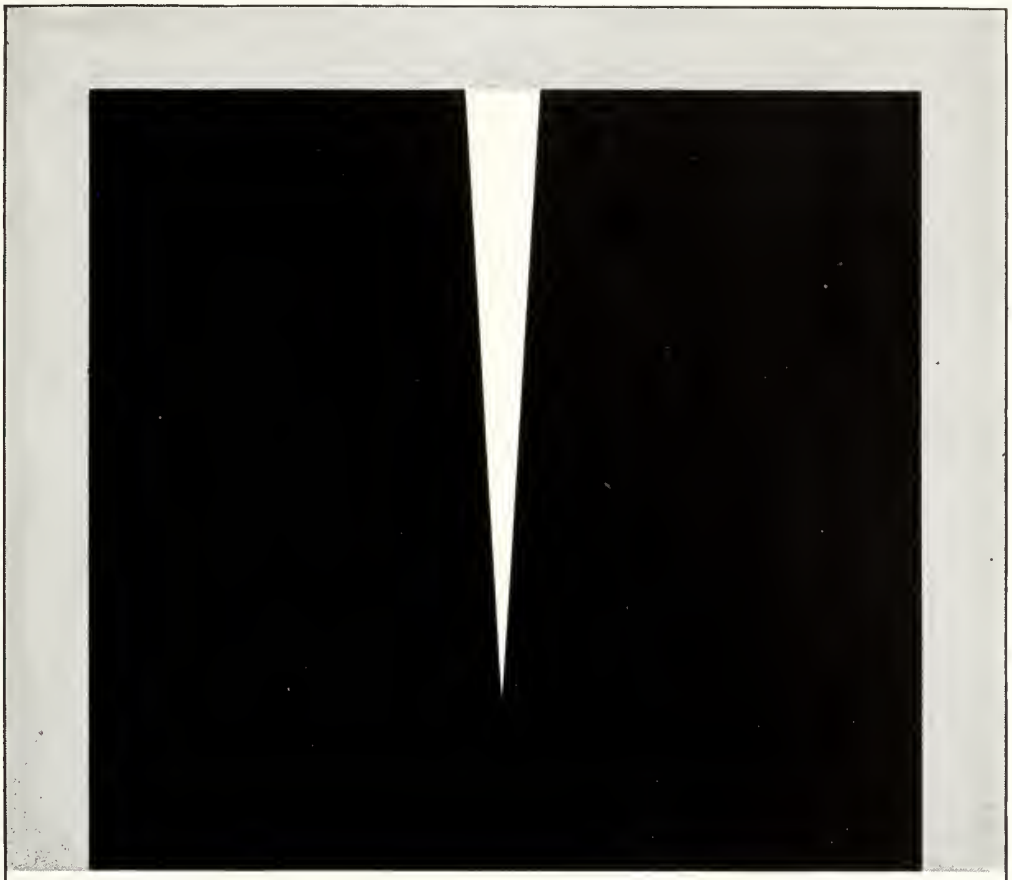
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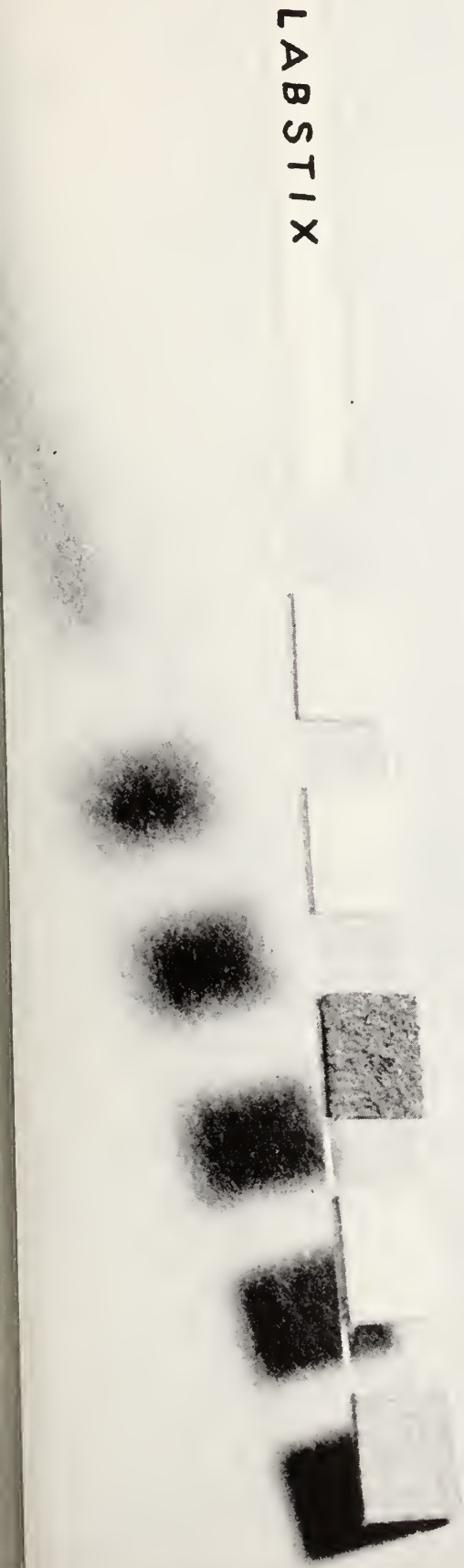
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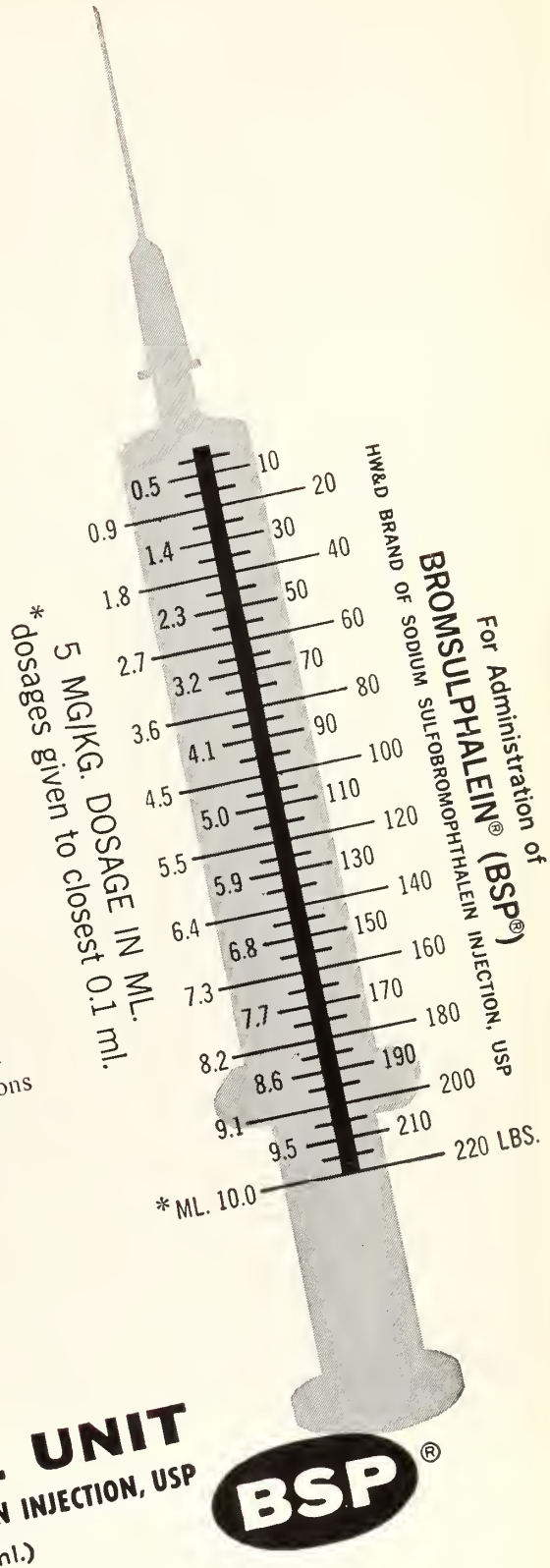
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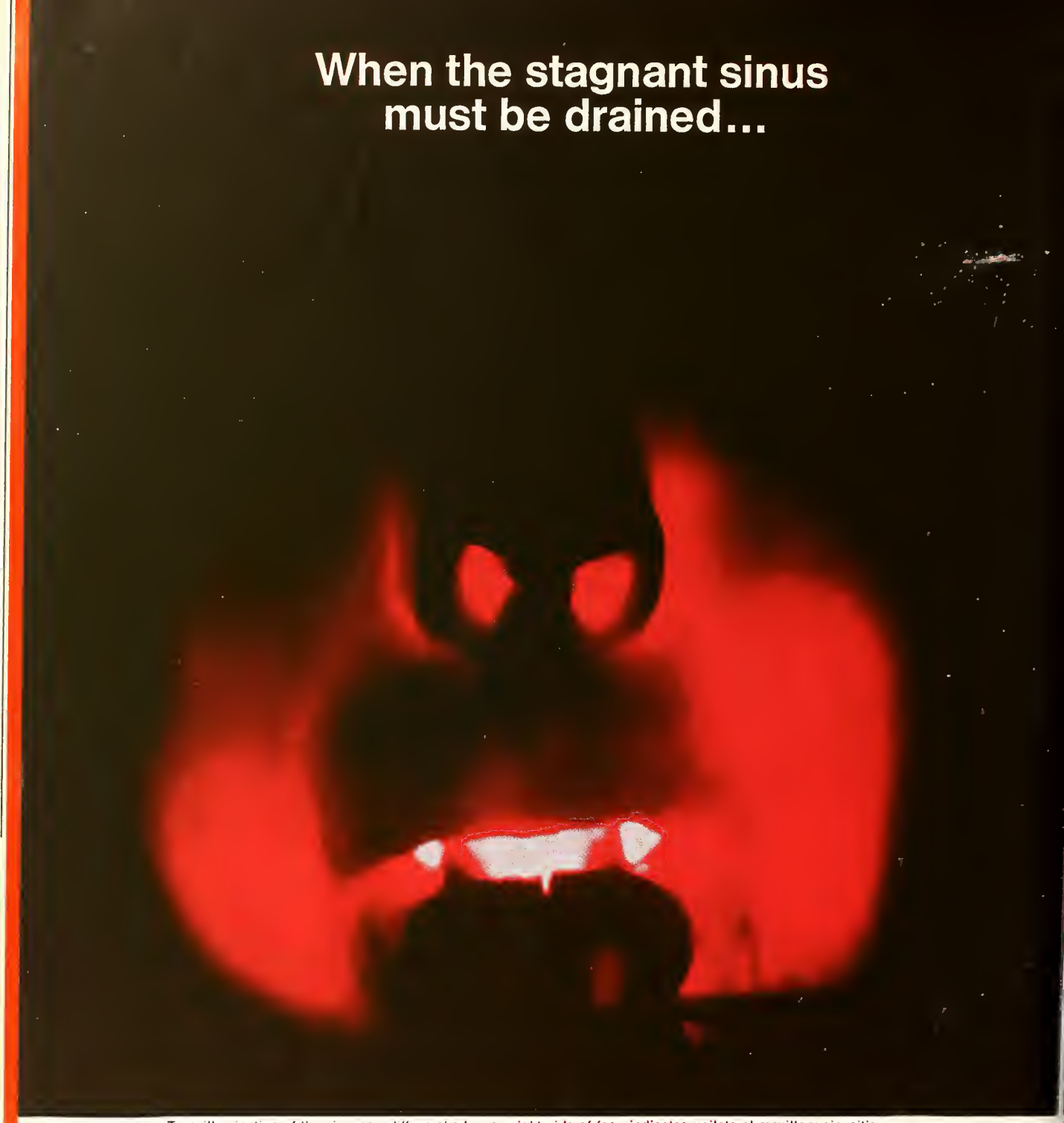
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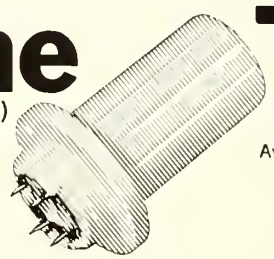
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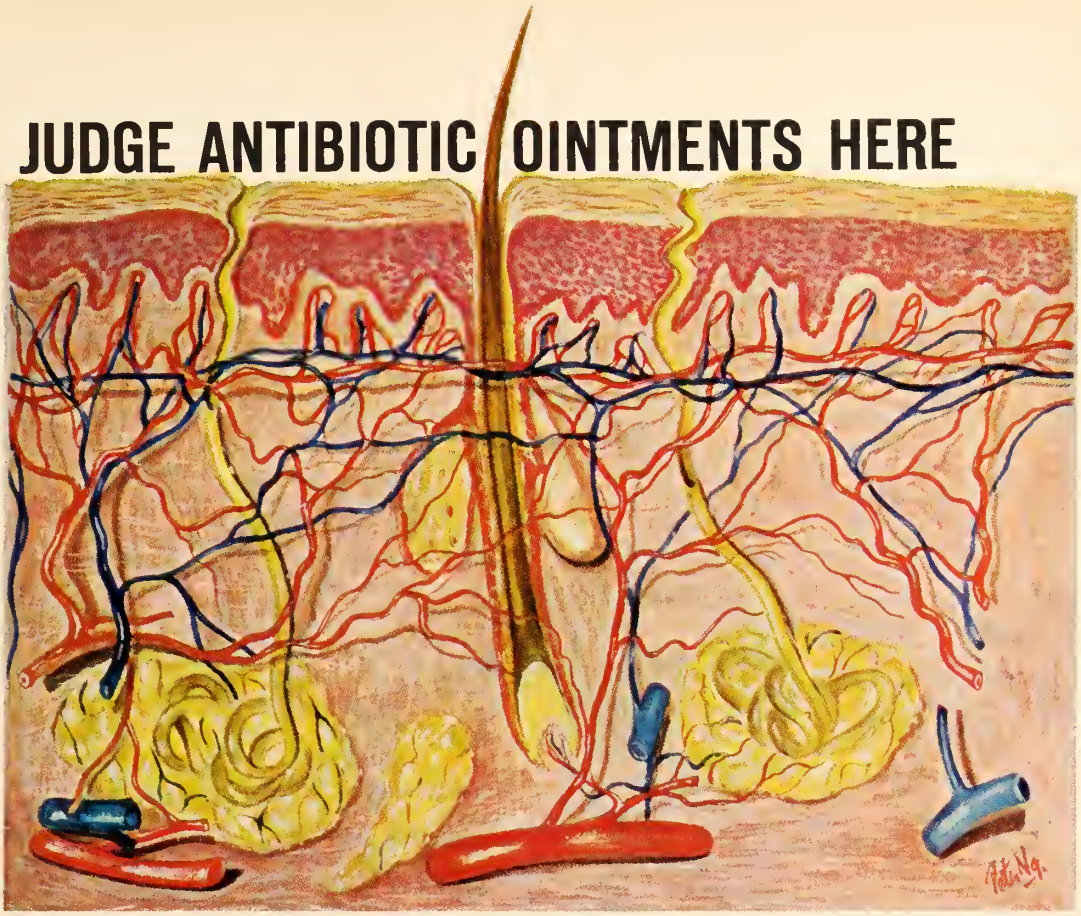
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Caution: May be habit-forming. Do not use in patients sensitive to barbiturates or in those with moderate to severe hepatic disease.

Side Effects: Nausea, transitory dizziness, rash. Overdosage of allobarbital produces symptoms typical of acute barbiturate excess.

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*Multiple of adult Minimum Daily Requirement supplied.
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Indications: 1. Functional fatigue such as that often associated with: a depressing life experience or stressful time of life; advancing years; convalescence; limited activity or confinement. 2. Poor appetite and vitamin-mineral deficiency as they occur in: patients having faulty eating habits; geriatric patients who are losing interest in food; patients convalescing from debilitating illness or surgery.

Dosage: Adults, 1 tablespoonful; children (over 15 years old), 1 to 2 teaspoonfuls; children (4 to 15 years old), 1 teaspoonful. To be taken three times daily 30 minutes before meals.

Contraindications: As with other drugs with CNS stimulating action, Alertonic is contraindicated in hyperactive, agitated or severely anxious patients and in chorea or obsessive compulsive states.

Side effects: Reports of overstimulation have been rare. Patients who are known to be unduly sensitive to the effects of stimulant drugs should be observed carefully in the initial stages of treatment.

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MOLECULAR REMODELING—

laboratory exercise or clinical necessity?

More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors —in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9,10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

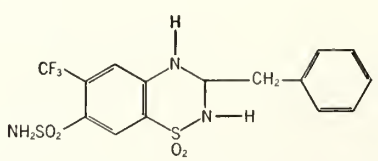
Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumethiazide a rather dramatic reduction in dose range is accomplished. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effective antihypertensive agent.



Contraindications: Severe renal impairment; previous hypersensitivity.

Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

References: **1.** Southworth, H.: Proc. Soc. Exper. Biol. & Med. *36*:58, 1937. **2.** Mann, T. and Keilin, D.: Nature *146*:164, 1940. **3.** Pitts, R. F., and Alexander, R. S.: Am. J. Physiol. *144*:239, 1945. **4.** Schwartz, W. B.: New England J. Med. *240*:173, 1949. **5.** Friedberg, C. K., in Moyer, J. H., and Fuchs, M.: Edema Mechanisms and Management, Philadelphia, W. B. Saunders Co., 1960, p. 259. **6.** Cumming, J. R.; Tabachnick, E., and Seelig, M., in Moyer, J. H., and Fuchs, M.: op. cit., p. 254. **7.** Werko, L., in Moyer, J. H., and Fuchs, M.: op. cit., p. 188. **8.** Beyer, K. H., Jr., in Moyer, J. H., and Fuchs, M.: op. cit., p. 274. **9.** Maren, T. H., and Wiley, C. E.: J. Pharmacol. & Exper. Therap. *143*:230, 1964. **10.** Earley, L. E., and Orloff, J.: Ann. Rev. Med. *15*:149, 1964. **11.** Fuchs, M., and Mallin, S. R., in Moyer, J. H., and Fuchs, M.: op. cit., p. 276. **12.** Ford, R. V., in Moyer, J. H., and Fuchs, M.: op. cit., p. 290. **13.** cited in Fuchs, M., and Mallin, S. R. (ref. 11): op. cit., p. 283.

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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

Contraindications: Patients hypersensitive to antihistamines. Not recommended for use during pregnancy.

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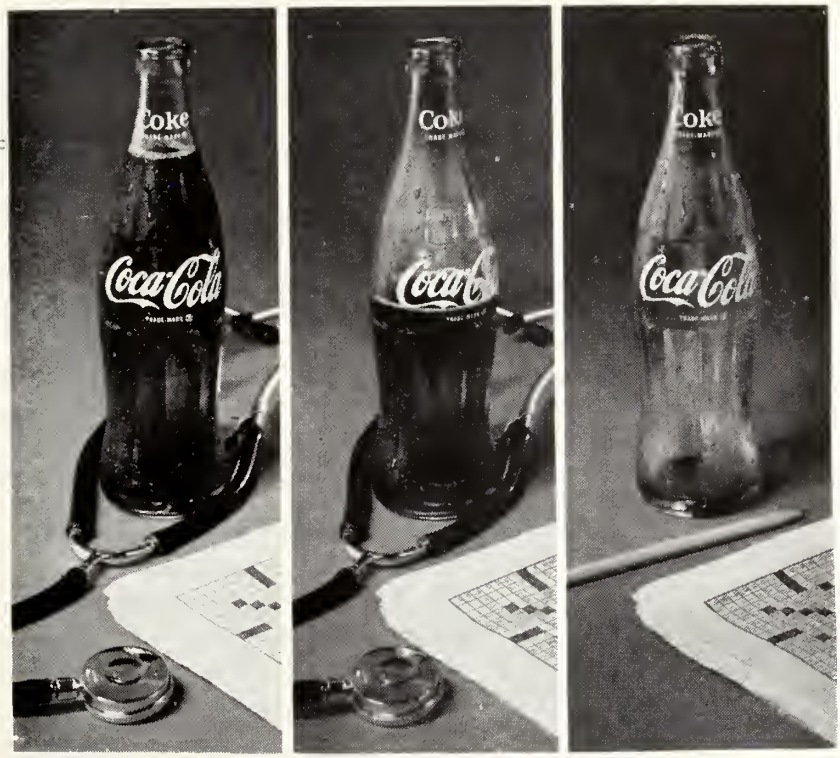
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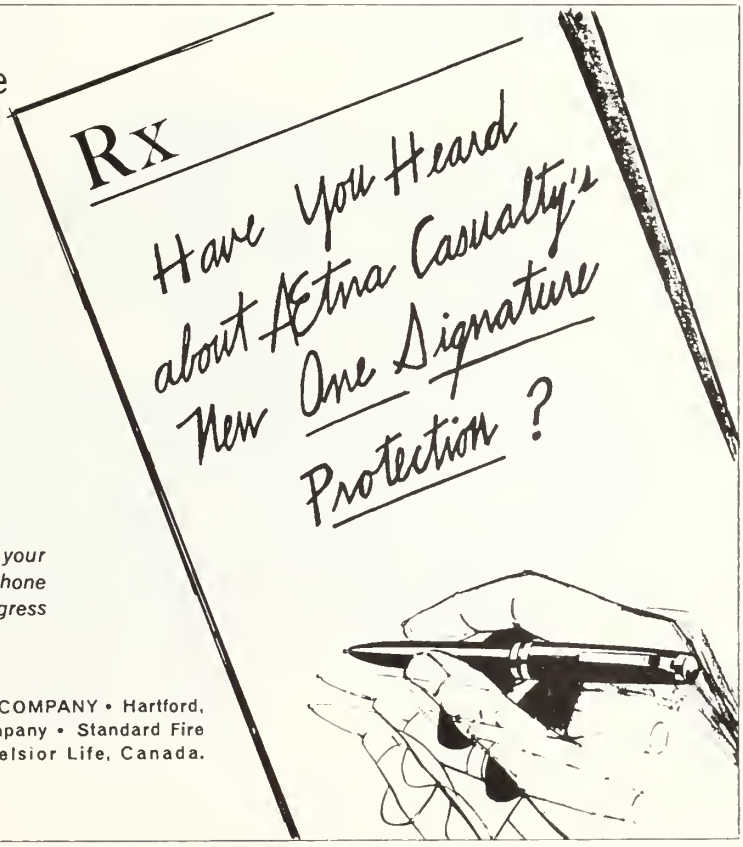
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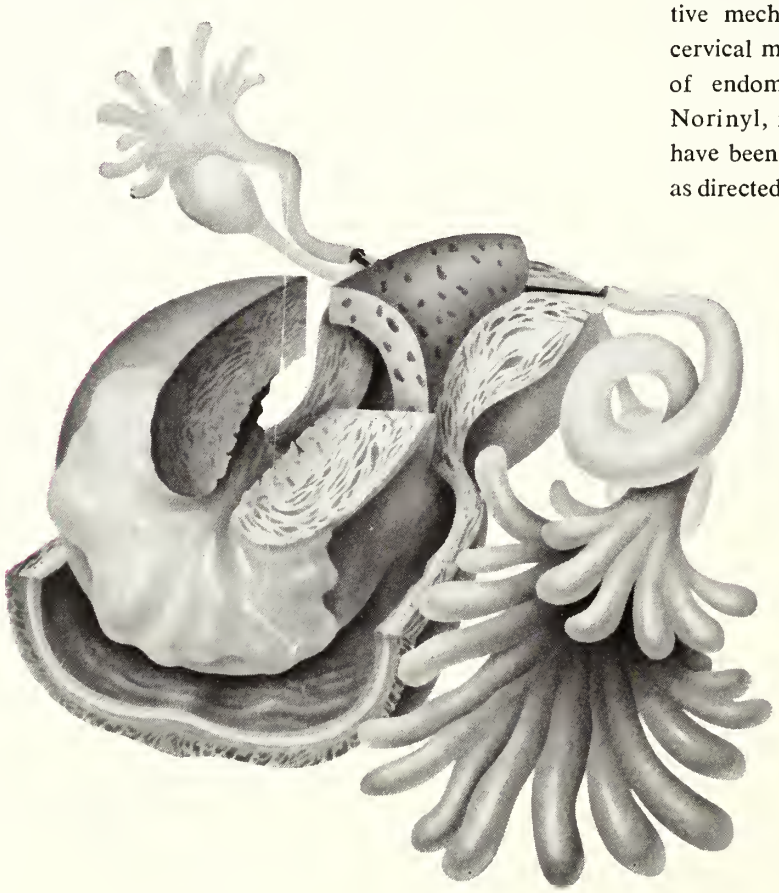
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inhibition of ovulation by means of 2 time-proved hormonal agents

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An unbreakable "confusionproof" package makes it easy to adhere to prescribed dosage schedule: individually sealed tablets numbered from 1 through 20 *plus* monthly calendar record enables patient to double-check dosage intake by day and corresponding tablet number.



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Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.–mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

References: 1. Council on Drugs. JAMA 187:664 (Feb 29) 1964. 2. Bryans, F. E.: Canad Med Ass J 92:287 (Feb. 6) 1965. 3. Goldzieher, J. W.: Med Clin N Amer 48:529 (Mar.) 1964. 4. Cohen, M. R.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965. 5. Hammond, D. O.: Ibid. 6. Rice-Wray, E., Goldzieher, J. W., and Aranda-Rosell, A.: Fertil Steril 14:402 (Jul.-Aug.) 1963. 7. Goldzieher, J. W., Moses, L. E., and Ellis, L. T.: JAMA 180:359 (May 5) 1962. 8. Kempers, R. D.: GP 29:88 (Jan.) 1964. 9. Tyler, E. T.: JAMA 187:562 (Feb. 22) 1964. 10. Rudel, H. W., Martinez-Manautou, J., and Maqueo-Topete, M.: Fertil Steril 16:158 (Mar.-Apr.) 1965. 11. Flowers, C. E., Jr.: N Carolina Med J 25:139 (Apr.) 1964. 12. Goldzieher, J. W.: Appl Ther 6:503 (June) 1964. 13. The Control of Fertility. Report adopted by the Committee on Human Reproduction of the American Medical Association. JAMA 194:462 (Oct. 25) 1965. 14. Flowers, C. E., Jr.: JAMA 188:1115 (June 29) 1964. 15. Merritt, R. I.: Appl Ther 6:427 (May) 1964. 16. Newland, D. O.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965.

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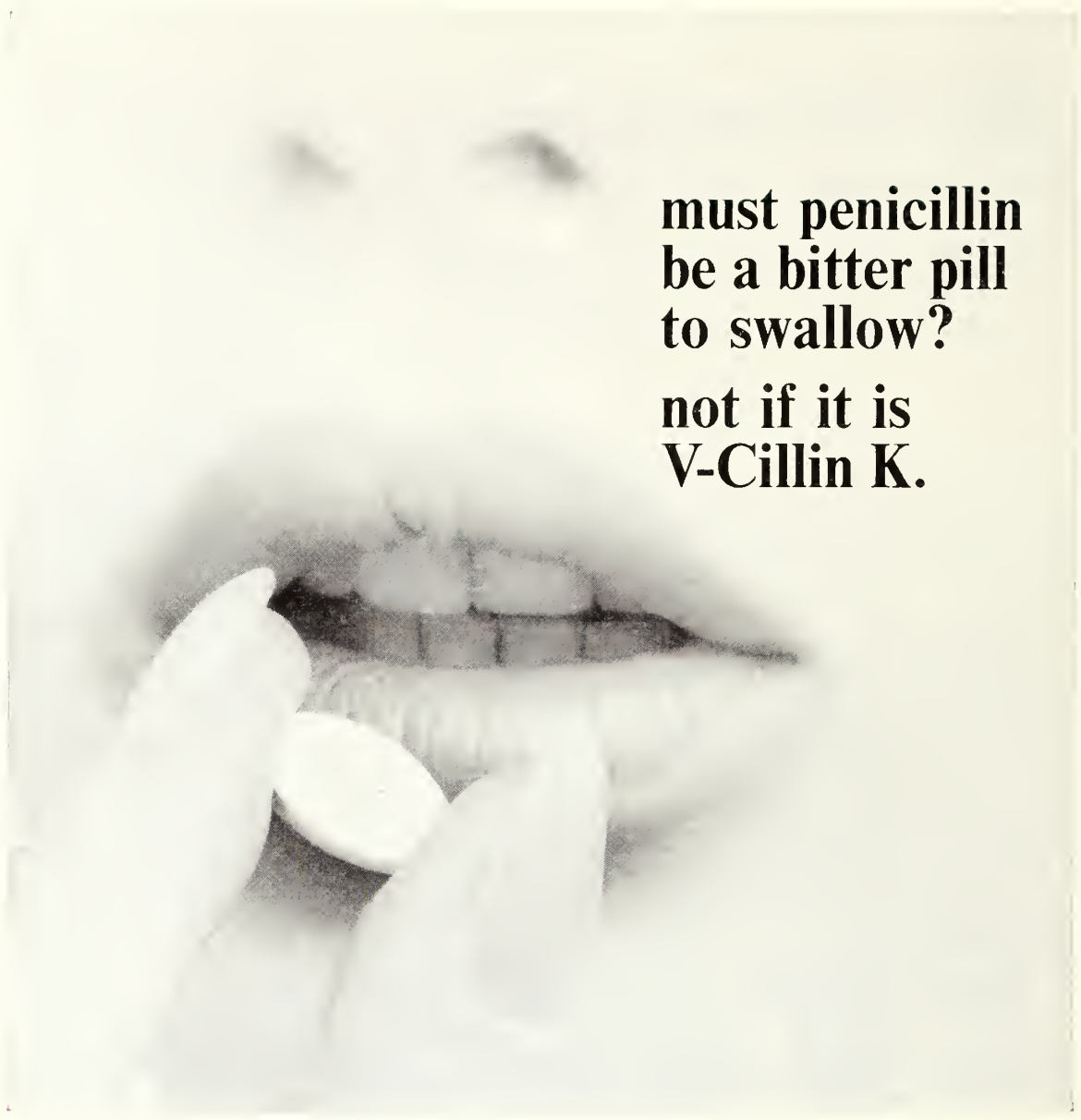
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Tuberous Sclerosis — A Study of Eight Cases†

J. Galindo, M.D.* and Elizabeth Beverage**

Tuberous Sclerosis, Bourneville's Disease, Epiloia, The Tuberous Sclerosis Complex, was originally described by Von Recklinghausen in 1862. He described patchy indurations of the cerebrum "tubers" in a newborn infant in whom he also encountered myocardial rhabdomyomas.

Bourneville[11] in a series of papers which he began publishing in 1880 gave this entity the name of tuberous sclerosis referring to the potato like tumors in the brain. His studies were continued by his associates Brisaud and Bannaire.

Balzer and Menetrier[3] were the first to describe the facial dermatologic component of the disease, specifically calling attention to the so-called sebaceous adenoma (a misnomer) whose classical histologic description was given by Pringle in 1890.[37]

Vogt in 1908[15] described the characteristic triad of cerebral tubers, mental retardation, epilepsy and adenoma sebaceum.

Sherlock in 1911[42] coined the term "epiloia" for this triad, a word picked for the peculiar reason that it was completely meaningless.

A number of possibilities concerning the nature of this process have been forwarded. The one which has received the widest acceptance is that of Moolten[32] who considers this process a disseminated hamartosis. As a result of the protean manifestations of the disease he suggested the term "Tuberous Sclerosis Complex" for the full blown syndrome of epilepsy, mental retardation, sebaceous adenoma, cerebral tubers.

Although originally only involvement of brain, skin, and heart were described, subsequently involvement of a myriad organs and anatomical sites has been described at one time or another.

Classically the cerebrum presents firm, flat, slightly depressed or slightly elevated nodulations (the tubers) which generally are easier to identify by palpation than by inspection.

Earlier authors suggested the inclusion of tuberous sclerosis within the group of the so-called Neuro Ectodermal Dystrophies; together with Von Recklinghausen's Neurofibromatosis; Sturge Weber's Disease and Von Hippel Landau's Disease; however, it must be noted that, involvement of tissues other than those of ectodermal origin may occur.

We have studied a group of 8 cases whose clinical and in instances autopsy findings are as follows:

Case No. 1

N.A.N., a male, was delivered at term with the use of low forceps on August 11, 1937. Peculiarities were first noted at 10 days of age when head jerking was noted by the mother; soon afterwards he began having grand mal seizures.

A review of the family history revealed no neurologic disorders in members of the paternal or maternal family.

Because of the clinical suspicion of a possible subdural hematoma a pneumoencephalogram was performed at 3 years of age; a temporal bone flap was elevated and ill delineated areas of induration became evident in the brain. The surgical incision was extended into the lateral ventricle in whose wall a number of knob like outcroppings were seen and biopsied. The skull x-ray revealed intracranial calcifications and a diagnosis of tuberous sclerosis was made. (Fig. 1)

In 1959 mushroom like phakomata were evidenced in the retina of the left eye. Because of mental retardation he was confined to a mental institution where he expired unexpectedly in July, 1963.

At autopsy a number of pinkish red, firm, closely grouped nodular elevations were present on the nasolabial folds, cheeks and ears.

The kidneys had adherent capsules and showed a number of pink to pink-white fairly well delineated nodules in cortex and medulla, the latter resulting in deformities of the papillae. (Fig. 2)

Histologic examination of these renal tumors revealed a diversified pattern on different sections consisting of arterioles

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Fig. 1. Irregular sized calcifications occur within the brain in a patient with tuberous sclerosis. Silver clips utilized in a previous surgical procedure are evident.



Fig. 2. Firm yellowish nodular hamartomata with poorly demarcated margins and absent capsules occur in renal cortex and medulla.

and venules of varied size with hypercellular walls from which appeared to arise smooth muscle cells that intertwined with similar adjoining cells forming small irregular bundles and faintly discernible curlicues. In addition there were two admixed types of cells within the tumor some of which had small round nuclei and others a large vesicular nucleus and small cytoplasmic rims. They formed a somewhat loose disheveled network among the smooth muscle like cells. Here and there one encountered groups of plump, round, fat cells with no identifiable abnormal features.

The pancreas showed one well demarcated tumor without true capsule (Fig. 3) which was composed of a somewhat monotonous picture of small and large cells with basophilic rounded and vesicular nuclei and pale pink poorly defined cytoplasmic boundaries. This pattern was interrupted at intervals by vessels with thickened walls. Once in a while a large cell with more than one nucleus occurred. The suprarenal

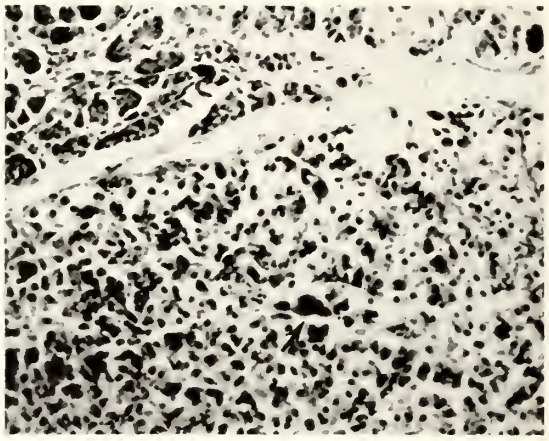


Fig. 3. Section of pancreas with normal acinar pattern seen at top and a sea of monotonous, epithelial cellular elements and vessels at bottom. Within the hamartoma are a number of bi- and tri-nuclear cells. (arrow) H and E X 150.

glands showed replacement of approximately 60% of their totality by nodular hamartomas somewhat reminescent in their histology of those seen in the pancreas.

Grossly the lungs did not appear to have tumor, however, histologically there were numerous small arterio-venous-neural tumors (glomangiomata) which were the subject of a separate publication.[2] (Fig. 4)

In the skull evidence of the previous craniotomy was seen. In the right paracentral region there was a depressed scar, presumably where the ventricle had been surgically entered. There were a number of ill defined firm nodules in the ventricles which on cut surface were gritty to section, they occurred in the caudate nucleus and at different points in the ventricular wall resulting in obstruction of the left foramen of Monroe and consequently in hydrocephalus.

A number of white nodules occupied both cortex and sub cortical white matter obliterating within their confines the limits of the cortical festoon. Histologically there was an admixture of large and bizarre neuronal type cells and abnormally large astroglial elements.

Case No. 2

E.M., a female, born November 1, 1938 at full term, normal delivery, weight 7½ lbs. At age 4 months she suffered an upper respiratory infection and at about this time she began having convulsions. She sat unsupported at 2 years, 9 months, spoke at 3½ years and walked with support at 5 years. Due to her mental retardation she was confined to a mental institution.

In 1960 the diagnosis of tuberous sclerosis was made. She developed large, firm, cauliflower shaped tumors on both sides of the nose (Fig. 5) which were surgically removed with rather appealing cosmetic results.

A pneumoencephalogram in 1960 showed dilatation of the lateral ventricles more marked on the left with both lobes displaying Penfield's phenomenon and there were scattered calcifications.

Approximately 30 days before death she developed a low grade fever; her urine showed albuminuria 100mg%, and a number of granular casts, N.P.N. was 175mg%, CO_2 15 mEq/L, Potassium 3.2. She expired rather suddenly, one month after the initiation of this illness.

At autopsy linear surgical scars were evident where the blastomatous hamartomas had been removed. Lateral to the scar were a number of whitish pink, firm nodules up to 3mm. in diameter with similar nodules also evident on the eyelids.

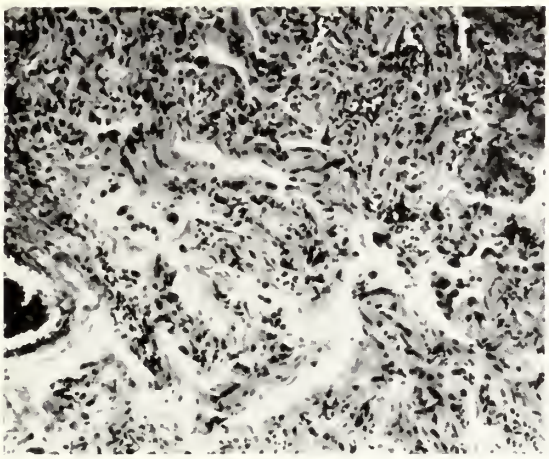


Fig. 4. Pulmonary glomangioma in a patient with tuberous sclerosis. With permission of Archives of Pathology, Vol. 78, pp. 287-291, September, 1964. H and E X 150.



Fig. 5. Blastomatous hamartomatas in paranasal areas. A number of smaller hamartomata are scattered on forehead and cheeks.

Confluent patches of bronchopneumonia occurred bilaterally. The heart had a number of ill defined soft, yellow areas which grossly were suggestive of rhabdomyomas but microscopically proved to be histologically unremarkable adipose tissue.

The stomach was enormously distended with gas and thin greenish fluid; its wall was thin and it extended caudally to the level of the symphysis pubis displacing the transverse colon in its path. (Fig. 6)

The kidneys were studded with well defined, soft and firm, yellow nodules which occurred both in cortex and medulla. Histologically they were richly vascularized hamartomas with scattered patches of closely grouped fat cells and irregularly scattered elongated myoid type cells.

The brain showed firm, poorly bounded zones of induration,

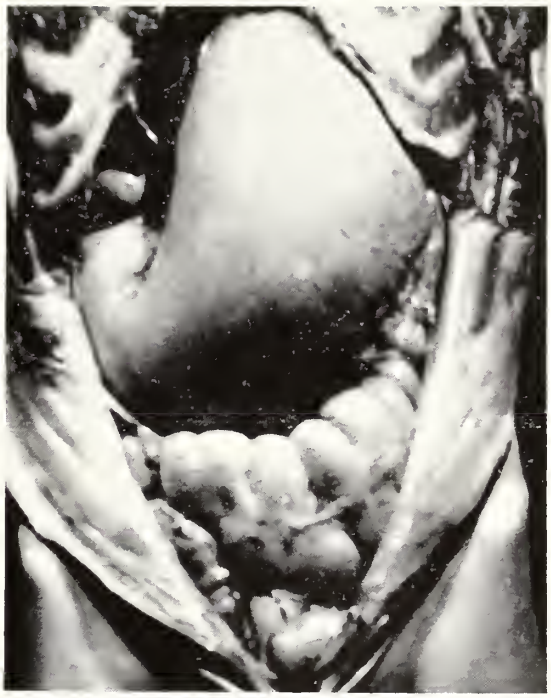


Fig. 6. Acute gastric dilatation with caudal displacement of the transverse colon in a patient with the tuberous sclerosis complex.

some slightly elevated, others depressed, corresponding to Pelizzi types I and II. (Fig. 7) The lateral ventricles were dilated but the third was not. Nodular excrescences were evident on the ventricular walls (candle gutterings).

Microscopically one encountered dysgenetic neurons and abnormally enlarged astrocytes in the involved cortex. (Fig. 8) Within the ventricular nodules composed of elongated cells in irregular pattern there were many calcareous deposits of varied shapes.

An interesting feature of this case and one that was the subject of a separate publication[1] was the terminal occurrence of an acute meningo encephalitis.

Case No. 3

A.T., a male, was delivered at full term, August 2, 1949, after labor of 3 hours duration, birth weight 8 lbs. He appeared normal initially till the age of 2 months when he began having convulsions. It became evident within the first 12 months that he was mentally retarded.

In 1959 a diagnosis of bilateral polycystic kidneys was made after retrograde pyelography. This same year for the first time dermal nodulations were noted on the face. One of these lesions was biopsied and was histologically classified as a Pringle's Nevus.

Radiography of the cranium demonstrated a number of intracerebral calcifications.

He was admitted to a mental institution where he expired in 1964 after a brief illness.

At autopsy the lungs showed bronchopneumonia. The brain showed cortical tubers and ventricular tumors were present in both lateral ventricles. (Figs. 9 and 10) The paranasal nodules were small and pinkish red, for the most part quite firm.

The kidneys were approximately three times normal size, the capsules were adherent and the normal gross architecture was totally replaced by numerous closely grouped cysts filled with clear yellow fluid. Neither grossly nor microscopically were hamartomata seen within them.

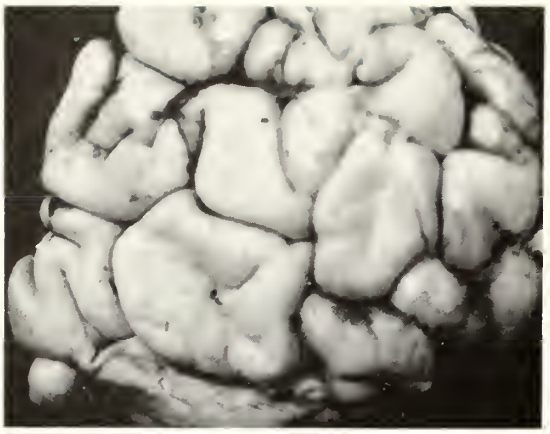


Fig. 7. Cerebral tubers depicting Pelizzi types I and II.

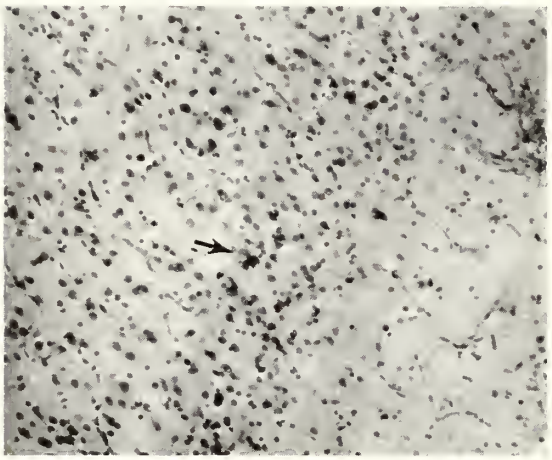


Fig. 8. Dysgenetic nerve cells some with more than one nucleus occur within a cerebral tuber. H and E X 150.

CASE NO. 4

P.G., born at full term, 1949 weight 8 lbs., 4 oz. At age 3 months he began having grand mal seizures. The electroencephalogram at age 7 months showed continuous high voltage, slow activity and occasional spikes.

He could not stand unsupported till age 3 years and could not dress himself till age 5 years. At about this time he began suffering from severe headaches which usually resulted in his having uncontrollable fits of temper.

On the Vineland scale he attained an S.M.A. of 1-9 and a Social Maturity Quotient of 36.

Because of the continuing severe headaches the clinical suspicion of increased intracranial pressure was raised and confirmed. A left frontal craniotomy was performed and a nodular mass which obstructed the left foramen of Monroe and dilated the ventricle was removed and was histologically diagnosed as an ependymoma.

He was admitted to Pineland Hospital and Training Center in 1965 at which time he showed nodules in the skin of face, neck and ears. (Fig. 11) Skull x-rays did not reveal intracerebral calcifications. He is still alive, but confined to a mental institution.

CASE NO. 5

D.G., a male, delivered by mid forceps after thirteen and one-half hours of labor on March 13, 1949. L.O.A. position, weight 4 lbs., 1½ oz. He remained in an incubator for 3 weeks after delivery. Teeth erupted at 6 months. At 8 months

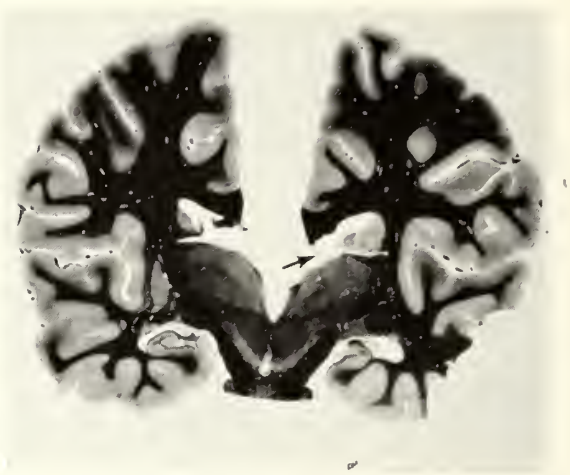


Fig. 9. Ventricular nodulations (candle gutterings) occur within caudate nucleus (arrow) Weil Weigert, giant celloidin section approx. ½ actual size.

he had his first convulsive seizure and has had a great many of them since. He walked at one year and began to speak at 2 years.

In 1964 he was classified as being in the severe range of mental retardation. Stanford Binet forms L and M revealed Mental Age of 3-8 and I.Q. of 22.

On physical examination numerous facial nodules were seen on the nose and the rest of the face. He had convergent strabismus. The deep tendon reflexes were unequal right vs. left and Babinski was positive on the right. Skull x-ray showed intracranial calcium deposits.

One day he presented with symptoms of acute abdomen; was operated on and a bleeding hamartoma was found on the right kidney which necessitated nephrectomy.[19] Since then he has returned to his pre surgical status and is confined to a mental institution.

CASE NO. 6

P.A.L., a female, second in order of birth, delivered with low forceps at full term, January 7, 1953, weight 7 lbs., 13 oz. She began having grand mal seizures at age 6 months which generally lasted less than five minutes and occurred several times in the course of a day.

Electroencephalogram revealed mild diffuse abnormality plus localized abnormality on the right mid cerebrum, particularly in parietal zone. Atrophy of right cerebral hemisphere was demonstrated after pneumoencephalogram.

She had left hemiparalysis with increased muscle tone on upper and lower extremities on the affected side. The left foot showed a severe inversion deformity.

On November 25, 1958 on the Stanford Binet Scale she attained a Mental Age of 2-11 with a Chronological Age of 5-10, I.Q. 50.

She slowly developed facial nodules on nasal and paranasal areas and is now confined to a mental institution.

CASE NO. 7

M.C., a male, born June 23, 1959 at full term, normal delivery, weight 7 lbs., 11 oz. He began having grand mal seizures at age 3 months which occurred 3 to 4 times a day. Sat unsupported at one year and walked at 18 months. He was never able to speak. Early in life it became evident that he was mentally retarded and he was confined to an institution.

At age 5 years his head circumference was 20 inches. He was unable to walk unsupported. He had an asymmetrical head with flat occiput. The patellar reflexes were hyperactive bilaterally,



Fig. 10. Low power view of a paraventricular nodule which shows the common pattern of elongated glial elements, calcospherites and scanty vasculature. H and E X 150.

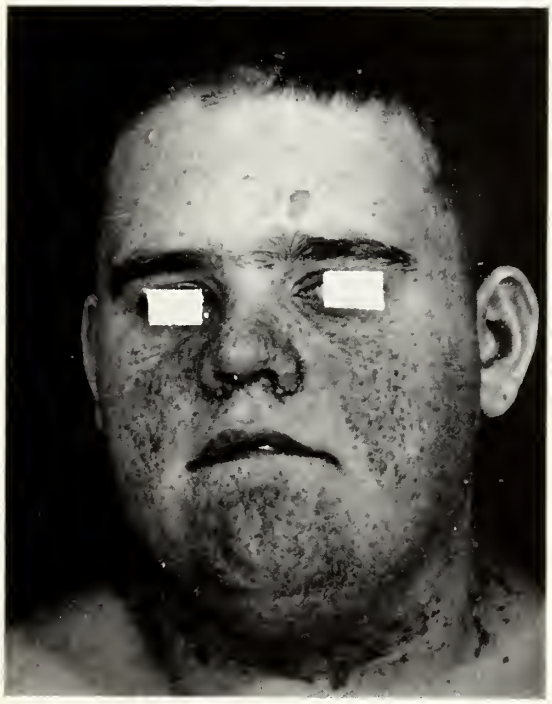


Fig. 11. Nodular fibrous hamartomata and adenoma sebaceum on nose, face and neck.

but more so on the right. There was a questionable Babinski on the left. There were no visible dermal nodulations at this time, but there were intracranial calcifications. Cytomegalic inclusion disease and toxoplasmosis were included in the differential diagnosis, but were excluded after extensive work-up. Apparently the patient has now developed facial nodules. He remains confined to a mental institution.

Case No. 8

C.E.M., a female, sixth in order of birth, delivered at full term, May 5, 1933 after prolonged labor. Birth weight 10 lbs. Teeth erupted at 7 months. She walked unsupported at one year.

The family history revealed no relevant data save for the fact that the maternal grandmother suffered from epileptic seizures.

At age 3 months C.E.M. began having grand mal seizures which occurred as many as 3 times a week. These seizures stopped rather abruptly at about age 6 years.



Fig. 11B. Fibrous nodules in typical peri-ungual location. Approx. 2 times normal size.

She learned to speak at approximately five years of age but always had difficulty in constructing sentences; most of her language consisting of disarticulated words and short garbled sentences.

Early in life it was recognized that she was severely mentally retarded and in 1947 she was placed permanently in a mental institution.

In 1955 when her chronological age was 21 years, 11 months she was tested on the Stanford Binet form L and obtained a mental age of 3 years, 3 months and an I.Q. of 20. She was retested in 1965 again using the Stanford Binet Test forms L and M and attained a Mental age of 2-11 and an I.Q. of 11. On the Vineland Social Maturity scale she attained a S.M.A. of 5-7 and a Social Maturity Quotient of 17.

In 1956 small dermal nodules appeared in the naso labial folds. In 1965 histologic study of one of these nodules was diagnosed as a Pringle's Nevus. She later developed periungual nodular fibromas. (Fig. 11B)

In October, 1965 her body length was 60½", Head Circumference 22". She had a high arched palate. The cranial nerves were intact and the deep tendon reflexes were normal.

Discussion

It seems reasonable at this time to emphasize that the full blown case of tuberous sclerosis with its characteristic features of epilepsy, mental retardation, Pringle's Nevi and intracranial calcification is not a difficult diagnosis to make. However, when one is confronted with the formes frustres in which only an isolated feature of the complex may be present, unless one carries a high index of suspicion the diagnosis may be difficult or at times impossible to make.

Mention has previously been made that Bourneville's Disease is inherited as a dominant trait with a high mutation rate. This appears well exemplified in the cases in which a number of family members are afflicted with the full blown complex, while others present only the formes frustre and still others remain unaffected.

Penrose[36] in his series of cases was unable to find affected relatives in half of them, a fact which led him to believe that in these the disease had probably arisen by mutation.

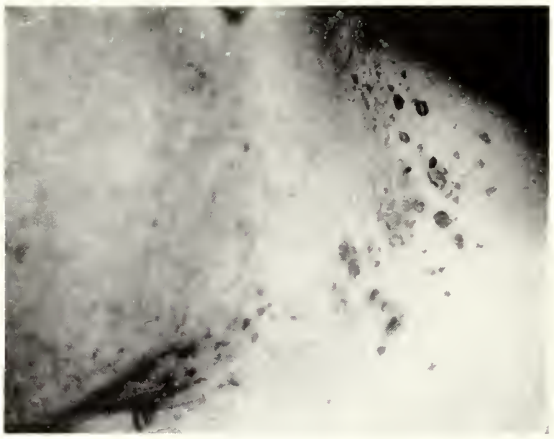


Fig. 12. Sessile and pedunculated hamartomata on the neck of a patient with tuberous sclerosis.



Fig. 13. Cerebral tuber within which giant astrocytes occur. Cajal Gold X 300.

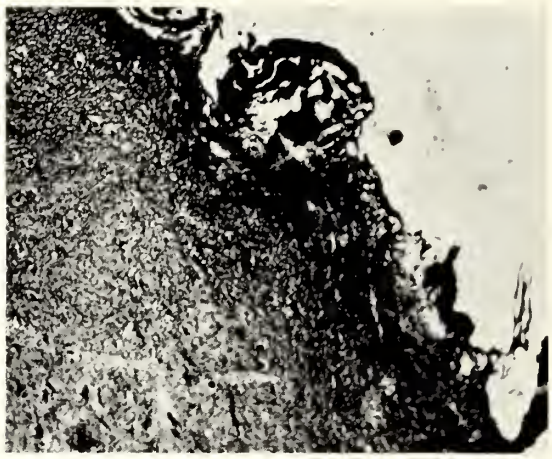


Fig. 14. A cortical tuber with neuronal dysgenesis and prominent gliotic peripheral festoons Holzer X 150.



Fig. 15. A loop-like gliomatous nodule projects into the lateral ventricle Holzer X 150.

We have already mentioned the organs which are most commonly affected in this disease, namely; brain, skin, myocardium and kidneys; however, infrequently other sites have been described; adrenals, pancreas, thyroid, breast, intestines and lung. The latter is one of the least common sites and it was not until 1938 that Berg[4] described the first case. One of our patients N.A.N. did present pulmonary involvement with the tumors being typical glomangiomas[2] (Fig. 4).

The bony involvement as described by Whitaker in 1959[16] may consist of sclerotic osseous changes which may be associated with bony cysts.

The close relationship of Von Recklinghausen's neurofibromatosis with tuberous sclerosis becomes evident, when one recalls that at times in the latter one encounters tumors of neurofibrillary nature and that in both diseases patches of dermal discoloration termed "cafe' au lait" spots occur.

Once in a while in addition to the milk and coffee dermal spots, areas of skin thickening occur particularly in the lumbar region which have been termed by some "shagreen patches".

Much has been written to date on the "sebaceous gland adenoma" of tuberous sclerosis, this term unfortunately widely used, is in fact a misnomer, for the sebaceous glands in the dermal nodulations are fully developed and are abnormal only in their number, Lever.[28] These nodules preferentially occupy the butterfly zone of the face. They normally are broad based, though in instances as was seen in one of our cases P.G. they may be attached by long pedicles. (Fig. 12).

These nodules are usually white, to pink white, brown or red, their coloration being dependent on their vasculature, and the relationship of the latter to the epidermis. They also show wide variation in their consistency from soft to hard, a factor related to the ratio of the connective tissue to other elements. Though most frequently seen in the face these nodules occasionally occur in ungual and sub ungual locations. (Fig 11B).

The brain usually is of normal weight though a case of tuberous sclerosis with micrencephaly was presented by Bielchowsky in 1923.[6] The contours of the brain are generally within limits of normality though one may see



Fig. 16. Myelin poor zones in an area where several tubers occurred. Weil Weigert Myelin stain approx. normal size.

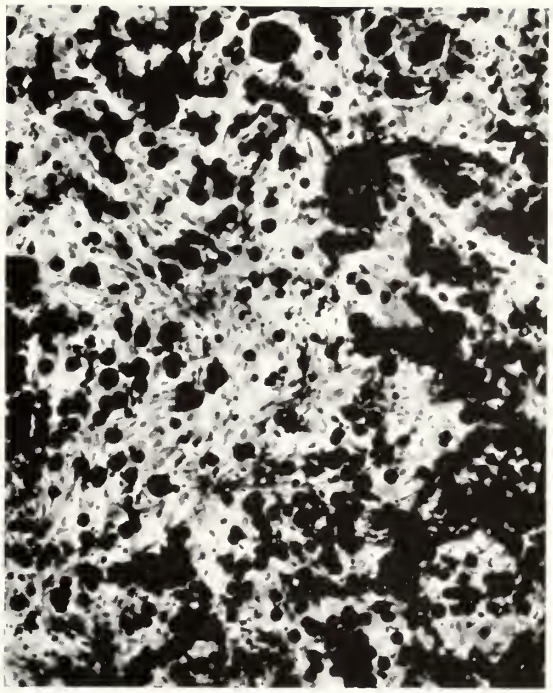


Fig. 17. Calcospherites, clubs and spicules in a paraventricular nodulation Von Kossa X 150.

widening and distortion of a gyrus with elevation or depression at the dome, or with shallow umbilication, circumscribed rounding and/or granularity corresponding to the types called Pelizzi 1 and Pelizzi 2. (Fig 7).

Characteristically there are hard tubers scattered in the cerebrum which are easier to palpate than to identify by inspection. These are generally white though they may have a faint yellowish tinge. Microscopically the tubers involve cortex or centrum semi-oval or both, in instances with loss of cortical boundaries. In these zones one encounters cells that have no specific arrangement; they are quite large often times exhibiting one, two or even three nuclei, prominent nucleoli and in instances vacuoles within the cytoplasm. A controversy still rages as to whether these cells are of neural or of glial origin. Certainly in the Cajal gold sublimate preparation one encounters many cells that are difficult to classify definitely as one or the other. (Fig. 13) They are probably a mixture of both.

In the Holzer preparation one encounters dense intermingled strands of fibrillary glia within the tubers. One of our cases showed a large irregular glial scar in the cortex (Fig. 14) while another showed a large glial nodule protruding into the ventricle. (Fig. 15) The Weil Weigert and Luxol Fast Blue stains show impoverished myelin staining in the tubers. (Fig. 16)

Within the ventricles and particularly within caudate nucleus nodular tumors are seen which when of adequate size and in the right location may obstruct the Sylvian aqueduct or the foramina of Monroe and result in hydrocephalus. (Fig. 9). These paraventricular nodules because of their characteristic aggregation have descriptively been termed "candle gutterings". Histologically they are composed of elongated or rounded cells with irregular nuclei which appear supported by fibrillary glia. (Fig. 10)

Calcifications are uncommon in cortical tubers, however, they become the rule rather than the exception in the ventricular nodules. These calcifications consist of concentric calcospherites and bizarre clubs and clusters which also stain positive for iron. When large enough these calcium deposits are an important feature of this process to the radiologist. (Figs. 1 and 17) An especially beautiful pattern is afforded by the Von Kossa stain on the calcified vessels within the nodules reminiscent of so many branches succumbing to the rigors of winter. (Fig. 18) These tumors are usually benign in nature, though occasionally malignant degeneration has been noted.

As we mentioned in the introduction the retinal phakomata were first described by van der Hoeve.[11] These flat or elevated and nodular tumors show a close kinship with paraventricular nodules, being composed of rounded or elongated poorly delineated cells and admixed glial elements. Calcification, and cystic degeneration within them is frequently encountered.

Pertaining to the renal nodules in tuberous sclerosis much has been written and little has been resolved. These tumors are generally well circumscribed whitish to yellow, devoid of capsules, varying in consistency

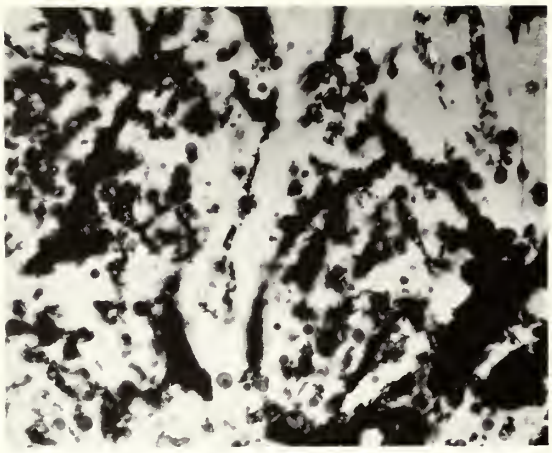


Fig. 18. Iron and calcium positive tree like vasculature in a paraventricular nodule. VonKossa and Prussian Blue X 150.

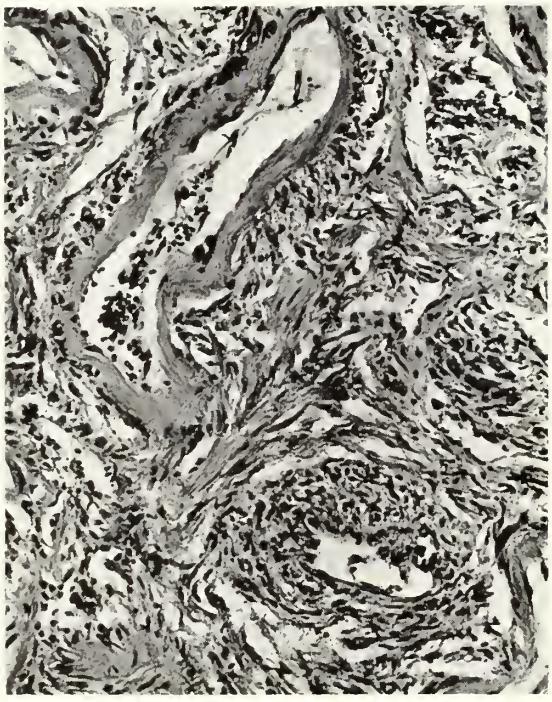


Fig. 19. Hypercellular vascular walls from which myoid elements appear to originate. Renal hamartoma in tuberous sclerosis H and E X 150.

from firm to soft. They are most frequently seen in the cortex though as seen in two of our cases 1 and 3 they may occur in the renal medulla. (Fig. 2) The variations in opinion center around their nature. Inglis for example considers them a distinct neoplasm whether they be associated with cerebral nodules or not. In his first paper in 1950[24] he suggests that their elongated cellular components might be of neurilemoblastic origin, in a later paper[26] he proposes the neural crest as their site of origin and still later[27] suggests that they are probably myoid in nature. In our cases the latter certainly appear to be the most likely possibility. However, these tumors

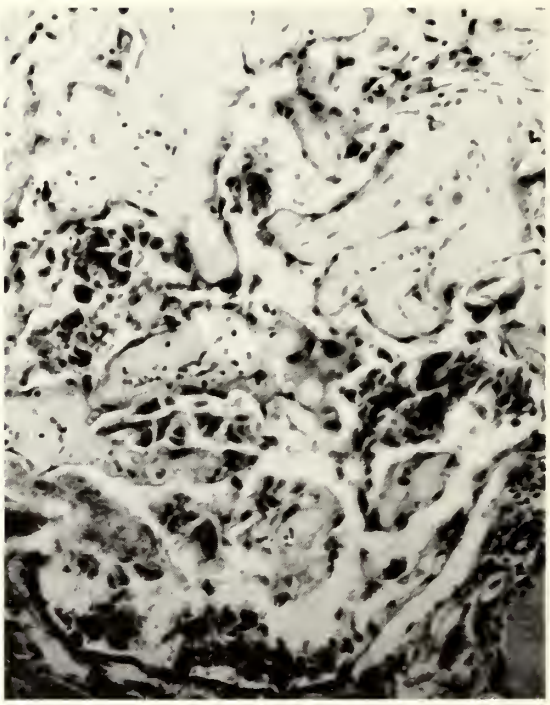


Fig. 20. Branchiogenic hamartoma replacing a parathyroid H and E X 150. With kind permission of Archives of Pathology, Vol. 78, pp. 287-291, September, 1964.

show quite an admixture of cells and variegated histologic pictures occur; it would be difficult therefore to assume that all have the same embryologic origin, though conceivably this might be the case. (Fig. 19)

The nature of the fat cells seen in the renal tumors is another puzzle. That the fat cells that occur in these tumors are indistinguishable from normal fat cells is indeed a fact; whether they originate from pluripotent cells or from cellular elements induced to store fat is at this time impossible to say.

The heart is frequently the site of single or multiple pale nodules that characteristically on histologic study show so-called spider cells which are rich in glycogen and though labelled rhabdomyomas would appear to arise from cells of the Purkinje system, therefore, being more deserving of the term Purkinjiomas. One of our cases No. 3 presented a number of pale yellow, soft, ill delineated zones within the myocardium which were grossly taken to represent rhabdomyomas, however, microscopically these consisted of areas of adipose tissue. We have been unable to find similar descriptions in other reported cases.

Pancreatic involvement was seen in one of our autopsied cases No. 1 (Fig. 3) which also showed replacement of one of the parathyroid glands by a branchiogenic hamartoma. (Fig. 20)

From the above we may conclude that tuberous sclerosis whether presenting as the full blown complex or as the formes frustres is indeed a hamartosis in which normally occurring elements present in bizarre and dishev-

eled patterns, and that the etiology of the process remains at present unknown.

Summary

Eight cases of tuberous sclerosis are presented, five males and three females, varying in ages from six to thirty-eight years. While the disease is inherited as a dominant trait it exhibits a high mutation rate. Some of our cases presented with the full blown tuberous sclerosis complex of epilepsy, mental retardation, intracerebral calcifications, and dermal nodules while others presented with only a few of the clinical features and represented formes frustres of this disease.

A review of the gross and microscopic characteristics of tumors in different organs encountered in our cases is presented.

Acknowledgment

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The Laurence-Moon-Biedl Syndrome†

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Laurence J. Z. and Moon, R. C.[17] in 1866 described four cases of retinitis pigmentosa in a family of ten siblings, two of whom had died early in life. Of these, three were males and one a female. They all presented visual difficulties which became more prominent under subdued light. Examination of the ocular fundi demonstrated a peculiar type of retinal pigmentation, consisting of small rounded or elongated spots from which fine dark lines radiated. Three of the patients were described as being of dull intelligence and two of them were obese. Polydactyly was not mentioned.

Bardet[3] and Biedl[5] described the complete syndrome consisting of failing vision, obesity, mental retardation, short stature and hypogenitalism. Raab[22] from Biedl's clinic emphasized the familial occurrence of this clinical entity.

In 1937 Warkany, et al[32] analyzed 102 cases of this syndrome again noting its recessive mode of transmission. Anderson[1] reviewed the autopsy records of six cases of the Laurence-Moon-Biedl Syndrome in which he noted an increase of pituitary basophils and Siegler[29] mentioned the occurrence of diabetes mellitus in the father and mother of the patient he described.

The complete syndrome of retinitis pigmentosa, failing vision, mental retardation, short stature, hypogenitalism, poly or syndactyly is well established. However, it is unfortunate that a number of cases in which only one of the symptoms occurred and there was no familial incidence, have crept into the literature. Therefore, of approximately 300 cases described, in our opinion not more than one hundred can definitely be included in this syndrome.

CASE NO. 1

A male, white, second in order of birth, delivered 10-8-55 at 7½ months gestation; weight 4 lbs., 15½ oz. Immediately after birth supernumerary digits were noted on all four limbs, which were subsequently surgically removed. Teeth erupted at 6 months; he began to walk unsupported at 22 months and spoke at 4 years.

The family history revealed no evidence of consanguinity. One maternal uncle had an extra thumb. Although no retinitis pigmentosa was evident in other family members examined, a remarkable number of them wore glasses.

Mental deterioration was noted early in life. In 1961 at the age of 5 years and 3 months he attained a mental age of 2 years and 2 months and an I.Q. of 35 on the Stanford Binet form L. He was evaluated twice in 1965 using form LM of the Binet test, and attained a mental age of 3 years, 6 months and an I.Q. of 37 in February and a mental age of 3 years, 7 months and an I.Q. of 36 in September. His social maturity was equally limited, as was indicated by a Vineland Social Maturity Age of 3 years, 2 months and Social Maturity Quotient of 34. He was classified as mentally retarded, severe, trainable level.



Fig. 1, Case No. 1: Hypogenitalism, obesity with presence of striae in arms and legs, short stature.

The electroencephalogram revealed an eye movement artifact with predominant rhythm of 8 per second alpha, which was most prominent in the occipital area. There was some scattered slower activity in the 3 to 4 per second range. The impression was of a borderline tracing without evidence of focal abnormality.

At age 9 years, he weighed 100 lbs.; had a body length of 53 inches (Figs. 1 and 2) and a head circumference of 20 inches. Blood pressure 132/78. He was obese, particularly around the thighs, abdomen and buttocks, and had a short neck and a high arched palate.

The right thigh was ½ inch smaller in diameter than the left. There was mild outward rotation of the hips, 4½ inches genu valgum, bilateral flare of the great toes and pes-planus with mild right pronation.

Both hands showed short digits and appeared spatulate (Fig. 3). The proximal phalanges were broad and the nails were wider than they were long. There were linear surgical scars at the site of amputation of the extra digits.

X-ray examination of both hands (Fig. 4) showed the fifth metacarpal to be longer than usual and at the base of the fifth metacarpal on each side there was a vestigial attempt at divi-

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Fig. 2, Case No. 1: Lateral view showing accumulation of adipose tissue on abdomen and buttocks.

sion for an extra digit, with evidence of surgery on the left. The bones appeared normal; however, the hands were squared and spatulate as seen in cases where a 6th digit is present. Bone age was consistent with the patient's age.

X-ray films of the feet (Fig. 5) showed the 5th metatarsal to be longer than normal, with division of its articulating distal head on the left showing the site of a previously developed 6th toe that had been amputated. X-rays of the skull were within normal limits.

Genitalia: The penis was small, circumcised. One testicle was in the scrotum and appeared normal to palpation. The other was undescended.

He wore thick glasses – 10.00 O.U. and suffered from failing vision particularly in subdued light. Retinoscope – 700-700. The vitreous was fluid with some opacities. The discs were pale, white-gray and the retinal vessels were attenuated. On the temporal side of the retina, there were several roundish, pigmented spots characteristic of retinitis pigmentosa.

Since he appeared to have auditory difficulties a Pure Tone Audiometry was performed with the following results:

AIR CONDUCTION I.S.O. 1964

	250	500	1000	2000	4000	8000
R.E.	45	45	40	45	45	40
L.E.	50	55	45	45	45	50



Fig. 3, Case No. 1: The fingers are short. The phalanges are widened. The nails are wider than they are long. Scars resulting from the surgical procedures are evident on the right hand.



Fig. 4, Case No. 1: The fifth metacarpal is long. At its base bilaterally there is a vestigial attempt at division for an extra digit.

BONE CONDUCTION I.S.O. 1964

	500	1000	2000	4000
R.E.	5	0	0	5
L.E.	5	10	5	5

The interpretation was definite impairment, air conductive loss, bilateral.

Laboratory examinations: Urine analysis and complete blood count were within normal limits. Fasting blood glucose (Somogyi-Nelson) 100mg%. VDRL – nonreactive. Blood type, O, cdE/cde, MN positive, Fy^a positive. Whole blood and urine chromatograms revealed normal amino acid patterns. P.B.I. – 6.1 micrograms per 100ml. Buccal mucosa – sex chromatin negative in 100 cells counted.

A short term leukocyte culture was undertaken employing a modification of the method by Moorehead, et al.[21] A total of 25 chromosomal spreads were counted and 3 were karyotyped (Fig. 6) with the following results:

Number of chromosomes:	45	46	47	48
Number of cells:	1	22	1	1

We were unable to detect either morphologic or other abnormalities in either autosomes or sex chromosomes. Study of the cells with 47 – 48 count did not show any consistent abnormality.

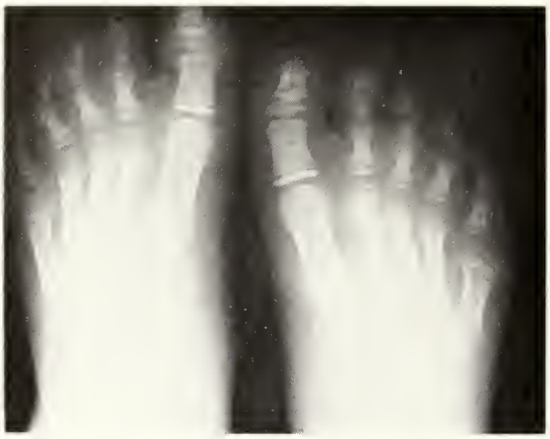


Fig. 5, Case No. 1: The fifth metatarsal is longer than normal. Division of the articulating distal head is evident.



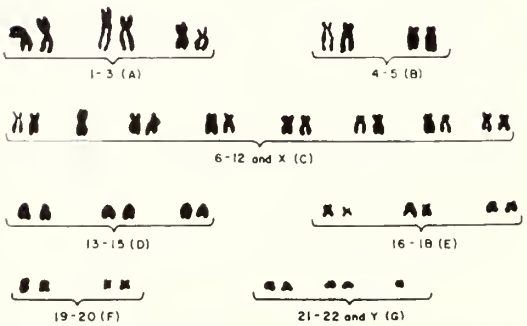


Fig. 6, Case No. 1: Chromosomal count of 46. Sex chromosomes are unremarkable. One autosome of pair No. 2 is slightly larger than its mate.

CASE NO. 2

(Fig. 7), A brother to propositus No. 1, fourth in order of birth. The mother had been admitted in false labor on two separate occasions, 5 weeks and 3 weeks prior to delivery, which occurred at full term on 1-17-59 weight 7 lbs., 11 oz. It was immediately noted that he had two extra digits on the right hand and one small extra digit on the left hand. On both feet there were six toes arranged quite symmetrically.

Three and a half months following delivery, he was readmitted to the hospital with a chief complaint of inability to gain weight, listlessness and absence of voice. Teeth erupted at 6 months. He could walk with support at 22 months and at this time could say "Mama" and "Dada." He could walk unsupported at 3½ years. He had measles and chickenpox before the age of 5 years.

In 1960 he underwent amputation and disarticulation of the right 6th toe.

Ophthalmoscopic examination demonstrated left esotropia and amblyopia with fixation nystagmus.

In October 1964, when he was 5 years, 9 months of age he



Fig. 7, Case No. 2: Obesity and hypogenitalism occur. There are a number of prominent subepidermal veins in the thorax and upper abdomen.



Fig. 8, Case No. 2: The digits are short and blunt. The fifth digit on the left is as wide as the thumb. The fifth finger bilaterally is curved towards the midline.

was given the Cattell Infant Intelligence Scale in a clinic for mentally retarded children. He attained a mental age of 2 years, 1 month and an I.Q. of 44. Evaluated again in February and September of 1965, using the Stanford Binet Form LM, he attained a mental age of 2 years, 7 months, I.Q. of 36 on the initial testing, and a mental age of 2 years, 7 months, I.Q. of 32 on the latter testing. His Vineland Social Maturity Scale yielded a Social Maturity Age of 3 years, 2 months and a Social Maturity Quotient of 47 at the time of the September evaluation.

Physical examination at 6 years of age: weight 55 lbs., body length 42½ inches, head circumference 19¾ inches, pulse 88, blood pressure 100/60. The general appearance was that of a moderately obese, active, cooperative child. The face displayed



Fig. 9, Case No. 2: The fifth metacarpal bilaterally is longer than normal. The middle and distal phalanx of the fifth digit are divided.

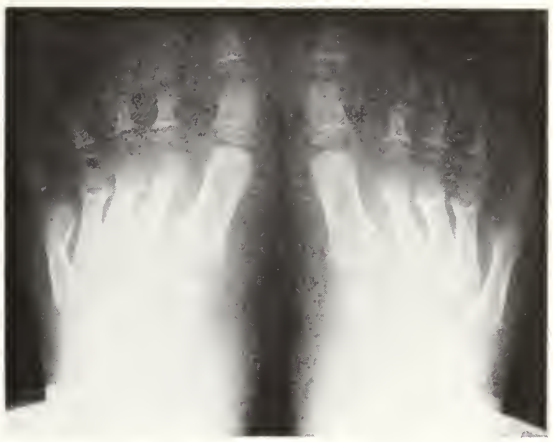


Fig. 10, Case No. 2: The first metatarsal, the proximal and the distal phalanges are short and broad. The phalanges of the fifth toes bilaterally are longer than usual and give the foot a squared appearance.

asymmetry; the neck was short with the head tending to tilt to the left. There was outward rotation of the hips and genu valgum was present. Digits of both hands were rather short and blunt, the distal phalanx giving the appearance of spade-like hands. The fifth digit on both hands was incurved and the one on the left was larger in diameter than the rest (Fig. 8). The genitalia were small, the penis being about one-half the expected size. The testicles were not palpable in the scrotum and he had enlarged inguinal rings. The cranial nerves were intact; the deep tendon reflexes were present and were equal bilaterally. Babinski and Romberg were negative.

The eyes showed round equal pupils that reacted normally to light and accommodation. There was internal strabismus on the left. Examination of the fundi revealed thin tenuous vessels and a moderate degree of atrophy. He had diminished vision and could distinguish large objects at 5 feet but not at 20 feet. Retinitis pigmentosa was minimal. Eso $\pm$ 40 Δ cross fixates Media clear. The discs were normal.

Laboratory examinations: Urinalysis and complete blood count were normal. Fasting blood glucose (Somogyi-Nelson) 95mg%, VDRL nonreactive, blood type O, cdE/cde, MN positive, Fy^a positive. Buccal mucosa – 2 cells in 100 showed a single sex chromatin body. Whole blood and urine one-way chromatography revealed essentially normal amino acid patterns. P.B.I. – 5.6mg/100ml.

X-ray examination of the hands (Fig. 9) revealed a fifth metacarpal that was longer than normal. The left middle phalanx and terminal phalanx of the fifth digit were divided and were fused at the proximal and distal ends. The diagnosis was that of congenital segmentation of the 5th digit.

X-ray examination of the feet (Fig. 10) showed the 5th toe to be somewhat larger than normal. The first metatarsal was larger, longer and wider than normal, giving added breadth to the foot.

Audiometry: Attempts to condition this child to pure tone audiometry proved fruitless. Informal clinical procedures were utilized in order to gain some knowledge of his auditory ability. He localized readily left and right, to voice at conversational level and noisemakers at 5 feet. Whisper at 10 feet was responded to quickly and appropriately. As a result of these gross informal methods it could be stated that he had acuity for daily routine, but, of course, it was not possible to state his threshold level.

Twenty-five cells from a peripheral blood short term leukocyte culture were studied and the following chromosomal counts were obtained:

Number of chromosomes:	45	46	47
Number of cells:	2	21	2

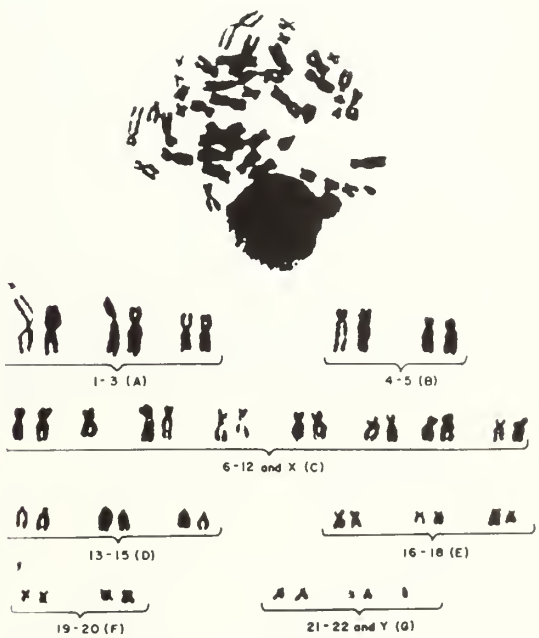


Fig. 11, Case No. 2: One member of each of the autosomal pairs 1 and 2 is slightly larger than its accompanying mate (this discrepancy in size was not consistent). The sex chromosome are morphologically not remarkable.

A total of 4 cells were karyotyped (Fig. 11) and no abnormalities were noted in either the autosomes or the sex chromosomes. Though in one of the preparations one of the autosomes in each of pairs No. 1 and 2 showed discrepancy in its size as compared with its mate.

Case No. 3

A white female, born 10-14-13, delivered by low forceps at full term after prolonged labor, weight 10 lbs., third in order of birth; no consanguinity. Teeth erupted at one year. She walked unsupported at 1½ years and was toilet trained at 3 years. At about this time she fell and lacerated her tongue so severely that she developed a speech impediment. She first went to school at ten years, but was unable to learn.



Fig. 12, Case No. 3: The fifth finger of the left hand is contracted, incurved and widened, at its distal end are two finger nails.



Fig. 13, Case No. 3: The proximal and distal phalanges of the fifth finger of the left hand are duplicated.

Physical examination in 1950 revealed an obese, white female with a body length of 5 feet, 7½ inches, blood pressure 140/90, weight 186 lbs., pulse 72. There was ptosis of the right eye, horizontal nystagmus, and strabismus on the right. The pupils were unreactive to light. Although it was not definitely established when she began having ocular difficulties, she was already complaining of severe visual impairment by 1927. By 1953, she was almost totally blind. At this time the deep tendon reflexes were hypoactive and the Babinski was negative. In 1965 her body length was 5 feet, 7½ inches, she was moderately obese. Joint range of motion demonstrated multiple restrictions with trunk extension, left shoulder external rotation. Right ankle inversion was severely limited. Mild restriction was present in trunk lateral flexion, hip flexion and abduction, knee flexion, shoulder extension, and wrist flexion and extension. There was no evidence of muscle atrophy but there was some weakness in the upper and lower abdomen and in the glutei.

The hands were broad and square with short stubby fingers. The right fifth finger was slightly incurved and the left little finger was contracted externally appearing as a double finger with two nails positioned side by side at its distal end (Fig. 12). X-ray of the left hand (Fig. 13) showed the fingers normally formed except for the 5th, which had a proximal phalanx widened at its distal end with a double articulation and a complete middle and distal phalanx which were incurved towards the midline.

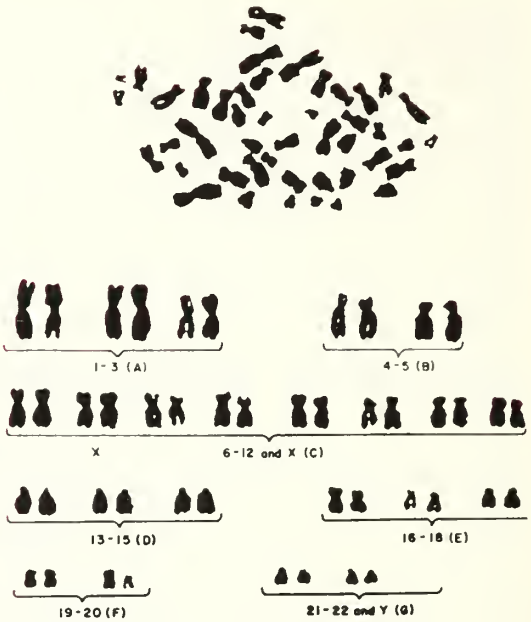


Fig. 14, Case No. 3: Normal karyotype.

The feet were blunt and squared with the great toes curved outward. She had four inch genu valgum and bilateral pesplanus.

The abdomen was moderately protuberant. The breasts were small and underdeveloped. Pubic hair was scanty and the labia majora were small.

The obesity was unequally distributed being particularly prominent in thighs, buttocks and lower abdomen. Heart sounds were normal, blood pressure 140/80.

Between 1925 and 1935 she was psychologically evaluated nine times with the revised Binet test. The initial testing yielded a Mental Age of 6 years, 5 months and an I.Q. of 53. At this time her chronological age was 12 years, 3 months. The subsequent evaluations showed a general decline in both Mental Age and I.Q. with the last evaluation in the series yielding a Mental Age of 5 years, 5 months and an I.Q. of 34. The decreasing Mental Ages and I.Q. were primarily a result of her failing vision. In 1958 an attempt was made to test her with the Wechsler Adult Intelligence Scale. Her blindness made it impossible to complete the performance portion of the test, although she attained a verbal I.Q. of 57. In August 1960, at the age of 46 years, 11 months, she was given the Hayes-Binet Test for the blind and attained a Mental Age of 7 years, 4 months and an I.Q. of 51. This was considered to be a valid assessment of her level of intellectual functioning and she was classified as Mentally Retarded, Moderate.

Electroencephalogram showed low voltage with beta activity anteriorly and a very persistant low voltage 3-5 per second slow waves and some anterior eye movement artifact. Activation procedures did not seem to change the record appreciably. The impression was that of a mildly abnormal record with diffuse low voltage slowing which was nonspecific and was consistent with diffuse encephalopathy.

Audiometry: Pure tone audiometry was done (Air Conduction I.S.O., 1964) Screening was done at 25db. for 250, 500, 1000, 2000, 4000, 8000 cycles. Her responses were clear-cut and valid. She had adequate threshold for daily routine. The left ear at 6,000 and 8,000 cycles lateralized and crossed over to the right. A check by masking and bone conduction was not possible due to noncooperation.

Ophthalmoscopic examination: Intraocular tension could not be determined due to the patient's uncooperativeness. However, on palpation the intraocular tension appeared normal. The vis-

ual fields showed generalized constriction, so that there was only a small central area of vision.

There were central, posterior, cortico-subcapsular, star shaped, opacities in the lens. The pupils were dilated and the fundi showed large variously shaped pigmentary deposits scattered over the retina. The central area of the fundi was not visible because of the cataracts. The impression was: 1. Retinitis pigmentosa, 2. Bilateral cataracts, 3. Nystagmus.

Laboratory Data: The CBC was within normal limits as were bleeding and clotting times and clot retraction. Fasting blood glucose (Somogyi-Nelson) 80mg%, sodium 139.5mEq/L, Cholesterol total 200mg%, Cholesterol Esters 148mg%, potassium 4.5mEq/L, Chlorides 101mEq/L, CO_2 combining power 30mEq/L, Blood type B, CDe/CDe, MN positive, Fy^a negative. Buccal mucosa a single Barr body was present in 21% of the cells. A total of twenty-five chromosomes were studied and the counts were as follows:

Number of chromosomes:	44	45	46	47
Number of cells:	1	4	17	3

Two cells were karytoped (Fig. 14), no consistent chromosomal abnormalities were evidenced.

Electrocardiogram was within normal limits.

Discussion

We have mentioned earlier that since the original description of Laurence and Moon,[17] a number of cases (approximately 330) have been described in the literature. Within this group, one is dismayed to find the inclusion of clinical cases which only remotely resemble the original description of the syndrome. The limits of this syndrome are vaguely defined; therefore, one encounters the description of both, cases that are either incomplete or supercomplete. This state of affairs has lead authors such as Warkany[33] to suggest that the Laurence-Moon-Biedl Syndrome is constituted by a fortuitous combination of several heredofamilial deviations. Radner[23] caluculated statistically the possibility of the fortuitous occurrence of polydactyly, mental retardation and retinitis pigmentosa combined and arrived at the figure of two cases per million population.

We are of the opinion that the above small number of cases can not justify the belief of random occurrence of the L-M-B Syndrome. If one is to include additional symptoms such as obesity, hypogenitalism, etc., this ratio would be increased even further.

There appears to be a definite male preponderance in this syndrome. Blumel[7] reviewed 65 cases in 47 families in which 39 (60%) were males and 26 (40%) were females. Even though the majority of cases described have been in Caucasians; Indians, Negroes, etc. afflicted with this syndrome have also been described. There is a definite direct relationship pertaining to consanguinity and the transmission of this syndrome as is portrayed in the cases reviewed by Cockayne[11] where marriages in affected families had occurred between first and second cousins. The condition appears to depend on an autosomal gene or genes which show wide variation in their degree of penetrance and expressivity. To our knowledge in previous cases of this syndrome the Karyotypes studied showed no abnormalities. However, Alström, et al, quoted by Bowen[8] described a sex chromatin-positive male with typical features of the Laurence-Moon-Biedl Syndrome. This certainly was not the case in the patients we studied.

The complete form of this syndrome includes mental retardation, retinitis pigmentosa, visual difficulties, obesity, hypogenitalism, polydactyly with or without syndactyly and offers no difficulty in the diagnosis. One is on less stable ground when only one or two of the above mentioned symptoms is present. In cases in which only one of the symptoms is present, familial occurrence is a sine-qua-non in order to permit inclusion within this syndrome.

Eyes: One of the most frequently encountered features in the Laurence-Moon-Biedl Syndrome is retinitis pigmentosa, which consists of dark retinal spots that are either rounded or elongated and show peripheral linear streaking. These finely pigmented areas resemble a sprinkling of pepper. Though the spots may be encountered in different areas of the retina, they appear to have predilection for the perivascular zones. Narrowing and paleness of the retinal vasculature has been described as are lesions within the macula, Taylor[30]; Warkany[33] found microphthalmos; and Burn[10] described myopia. In the cases presented by Blumel[7] there was bilateral nystagmus and associated strabismus. Radner,[23] in one of his two cases, encountered slanting of the eyes as one sees in Down's syndrome. Ocular ptosis and cataracts were present in Bhagwat's[4] review as were night blindness and normal fundi.

Skeleton: Osseous pathology of diverse nature has been described in this syndrome which varies from increase in size of a single short bone, i.e., carpal or metacarpal, to bony duplication of digits and osteoporotic changes associated with the hypogonadotropic hypogonadism. Variations in the spacial interrelationship of osseous structures has also frequently been described, i.e., genu valgum, pes-planus. Certainly one of the more frequent skeletal anomalies is the presence of extra digits with or without associated syndactyly (as in two of our cases, 2 and 3). Usually, the supernumerary digit number is six per extremity; however, as many as thirteen digits to a hand or foot have been described. In a number of instances, the extra "digit" contains no bone and is constituted in its entirety of soft tissue.

High arched palate, as in one of our cases (No. 1) has been encountered by other authors Attiah and El-Gamal,[2] Scott and Johnson.[28]

Genitalia: The hypogenitalism of the Laurence-Moon-Biedl Syndrome represents one of the cardinal symptoms in this syndrome.

The testicles are generally small, show increased consistency to palpation and may or may not be undescended. Histologically there is atrophy of the seminiferous tubules with hyalinization. The germinal epithelium is scanty and disarranged, the intertubular connective tissue shows varying degrees of compactness and or fibrosis and the interstitial cells of Leydig are prominent. In instances, the germinal epithelial depletion is extensive and only the Sertoli cells remain.

Roth[27] was the first to study with detail the hypogonadotropic hypogonadism in the LaurenceMoon-Biedl Syndrome employing combined urinary hormonal estimations and testicular biopsies. The results obtained by him are difficult to assess since his patients lacked a number of symptoms of this syndrome, namely retinitis pigmentosa, mental retardation and polydactyly, and only one of the patients was obese.

With sound clinical reasoning, a number of authors have tried to correlate the hypogonadism with visible histopathologic hypothalamic lesions. The latter has in general been unrewarding. Anderson[1] reviewed six cases of the L-M-B Syndrome and in all of them found an increase of pituitary basophils.

Carbohydrate Disturbances: Pathologic alterations in carbohydrate metabolism consisting of elevated fasting blood glucose levels have been seen, by some, in patients afflicted with the L-M-B Syndrome; however, no consistency in this finding appears to exist. Glucose tolerance curves studied by Montanaro,[20] Lurie[18] and Wendt[34] have been reported as being normal.

Hearing: From 1938 to 1948 six families with the Laurence-Moon-Biedl Syndrome were described in which nine members showed deafness. One of our patients (No. 1) showed definite air conductive loss bilaterally.

Heart: A number of cardiac anomalies pertaining to the myocardium have been described. McLaughlin[19] in a review of nine cases culled from the literature found defective development of the aortic cusps, double right coronary and hypoplasia of the aorta, in the patients described by Riggs[25]; ventricular septal and pulmonary valvular stenosis, Blumel[7]; Brattgard,[9] Radner,[23] Ross[26]; biventricular hypertrophy was present in Francke's[12] case.

Our three cases revealed no abnormalities in their cardiac work-up.

Mental Retardation: Varying degrees of impaired mentation have been described in patients with the L-M-B Syndrome. In general most of the cases appear to fall in the moderate to severe range of mental retardation. The cases we have described were all mentally retarded.

Obesity: The unevenly distributed obesity associated with hypogenitalism may be related to the pituitary and hypothalamus.

Summary

Three cases of the Laurence-Moon-Biedl Syndrome have been described, two males and one female. Polydactyly, obesity, retinitis pigmentosa with visual difficulties were present in all of them. In addition, one of them had cataracts. The familial incidence of this clinical entity appears well exemplified with the occurrence in two affected males of the full blown syndrome and polydactyly in their maternal uncle.

Acknowledgment

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Rapid Chromatographic Detection of Amino Acid Abnormalities

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Much scientific turmoil has followed in the wake of Sir Archibald Garrod's now famous Croonian Lectures and his monograph on "Inborn Errors of Metabolism"[4] in which were presented his accumulated observations on albinism, pentosuria, alcaptonuria and cystinuria.

Just as half a century was necessary to prove Garrod's correct assumption that the accumulated homogentisic acid in alcaptonuria was related to the absence of an enzyme (homogentisic acid oxidase); so have other delays occurred in the falling of barriers within the terrain of metabolic abnormalities.

Garrod's work, neglected for so long a time, has finally occupied the scientific pedestal it so rightfully deserves.

From the original four diseases described by him, the growing list now encompasses more than one hundred abnormalities, and the list still continues to grow. The various metabolic aberrations related to "inborn errors" have been cataloged into some eleven separate categories.[6]

Phenylketonuria, first described by Folling,[3] is probably one of the most widely known and more extensively studied of the amino acid metabolic aberrations. Here the relationship of the abnormal gene to an enzyme and the resulting abnormal clinical picture first suggested by Garrod has been clearly demonstrated. In this pathologic entity the accumulated phenylalanine and its lack of conversion to tyrosine as a result of inactivity of phenylalanine hydroxylase can clearly be demonstrated and accurately measured. Phenylketonuria, however, is but one of a group of more than 30 pathologic entities characterized by the abnormal accumulation of one or more amino acids.

The advent of methods of mass screening for the detection of the metabolic errors of amino acid metabolism such as Guthrie's inhibition technique[5] have opened previously unexplored areas which have already reaped the benefits of early diagnosis and, where available and possible, of corrective measures with dietary treatment.

A simple chromatographic method for the detection of abnormal amino acids in urine and blood was first introduced by Efron and her co-workers in 1964.[2] We have rewardingly employed Dr. Efron's chromatographic technique in our laboratory for the past year and have incorporated a variant of our own for the rapid detection of sulfur containing amino acid elevations. Complete details for this chromatographic technique are given by Efron and co-workers;[2] we will, therefore, touch but lightly on the methodology of this valuable procedure.

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METHODOLOGY

Amino Acid Chromatography – Blood and urine sampling is obtained by single or multiple spot application with intermittent drying on a square of good quality filter paper. Adequate reproducibility is obtained when 20 ul urine with a specific gravity greater than 1.015, 40 ul urine with a specific gravity between 1.010 and 1.015, and 60 ul urine with a specific gravity less than 1.010 is spotted within a circle of 1 cm. diameter with intermittent air drying (if the latter is to be hastened a hair dryer is quite helpful). If desired, an hemoglobin pipette may be employed for the urine spotting. Blood is spotted as for the Guthrie test. Once the blood and urine specimens have been properly dried they have the advantage of easy mailability and utilization after considerable time.

From the center of the dry spotted zone and with the use of a regular paper punch, a disc is obtained which is then transferred to a suitably located perforation punched with the same instrument in a rectangle of medium flow rate chromatography paper. Specimens are set up in duplicate on separate pieces of filter paper which are then rolled in cylindrical fashion and held in place with a clip or staple at the top. The papers are transferred to chromatographic tanks containing butanol/acetic acid/water solvent (BuA) 12/3/5. The tanks are sealed at the top and the solvent is allowed to migrate up the paper for approximately 16 hours.

The dried chromatograms are stained with ninhydrin and isatin respectively. Ehrlich's reagent and diasotized sulfanilic acid applied over the former stains are necessary for specific detection of some of the amino acids; eg, hydroxyproline, citrulline, and histidine, as adapted from Smith[9] and suggested by Efron.[2]

Modified Nitroprusside Test – Homocystine migrates with approximately the same Rf in BuA solvent as arginine and serine which are normally present in chromatographically detectable quantities. It, therefore, is advisable to screen the urine for an elevation of sulfur containing amino acids concurrently with the chromatographic run.

The cyanide nitroprusside tube method for the chemical detection of cystine in urine has been in use for a long time.[1] We have modified this method so that it may be performed on the discs of dried urine samples on filter paper.

Reagents for this method are prepared as follows:

1. 5% sodium or potassium cyanide with 1% ammonium hydroxide in 70% ethyl alcohol.

2. 5% sodium nitroferricyanide in 70% ethyl alcohol.

For each 100 ml. of reagent, dissolve 5 gms. of dry chemical in 25 ml. of distilled water. Add slowly and with constant stirring 75 ml. of 95% ethyl alcohol. One ml. of concentrated ammonium hydroxide is added to the cyanide reagent. Both reagents are stable for approximately 30 days when stored in tightly stoppered glass bottles.

A small punched disc from the dried collected sample on filter paper is placed on a rectangle of medium weight chromatography paper. The paper is dipped for a moment in the ammoniacal solution of sodium cyanide, allowed to drip dry for 2 minutes and then dipped into the sodium nitroprusside reagent. The stained paper is placed on a white background and the results read within 1 minute. The appearance of a pink coloration is indicative of a sulfur containing amino acid concentration greater than 5 mgs%. Semi-quantitative values may be obtained by comparison with known concentrations of cystine or homocystine.

It is important that there be no evidence of albuminuria as the normal cystine content in protein will cause false positive results.

Results and Discussion

As we have mentioned earlier the simplicity with which the samples can be collected and the small amount of equipment necessary to perform the tests render this chromatographic technique ideal for screening of large population numbers.

We have screened in a little over 10 months a hospital population of over 1,300 mental retardates in our laboratory. Among the study group were 13 known untreated patients and 2 previously undiagnosed infants with phenylpyruvic oligophrenia who clearly demonstrated elevated levels of phenylalanine in both blood and urine. The estimated concentrations correlated nicely with the spectrophotometric method of LaDu[7] and the fluorometric technique of McCaman and Robins.[8]

Also encountered in this screening procedure were one case of cystinuria,[10] two cases of hyperprolinemia, and a number of other abnormalities which are currently still under investigation.

With the simplicity of the method and its relatively low cost, this should be an ideal method for mass screening.

Summary

A brief description of a rapid chromatographic amino acid screening program using whole blood and urine is presented. A modified procedure for the detection of increased levels of sulfur containing amino acids is explained in detail. The described procedures were used in our laboratory to screen over 1,300 institutionalized mental retardates.

The ease of sample collection, the simplicity of the method and its relatively low cost render this procedure ideal for mass screening.

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5-8 years	8 mg.	1 tsp. 4 times daily
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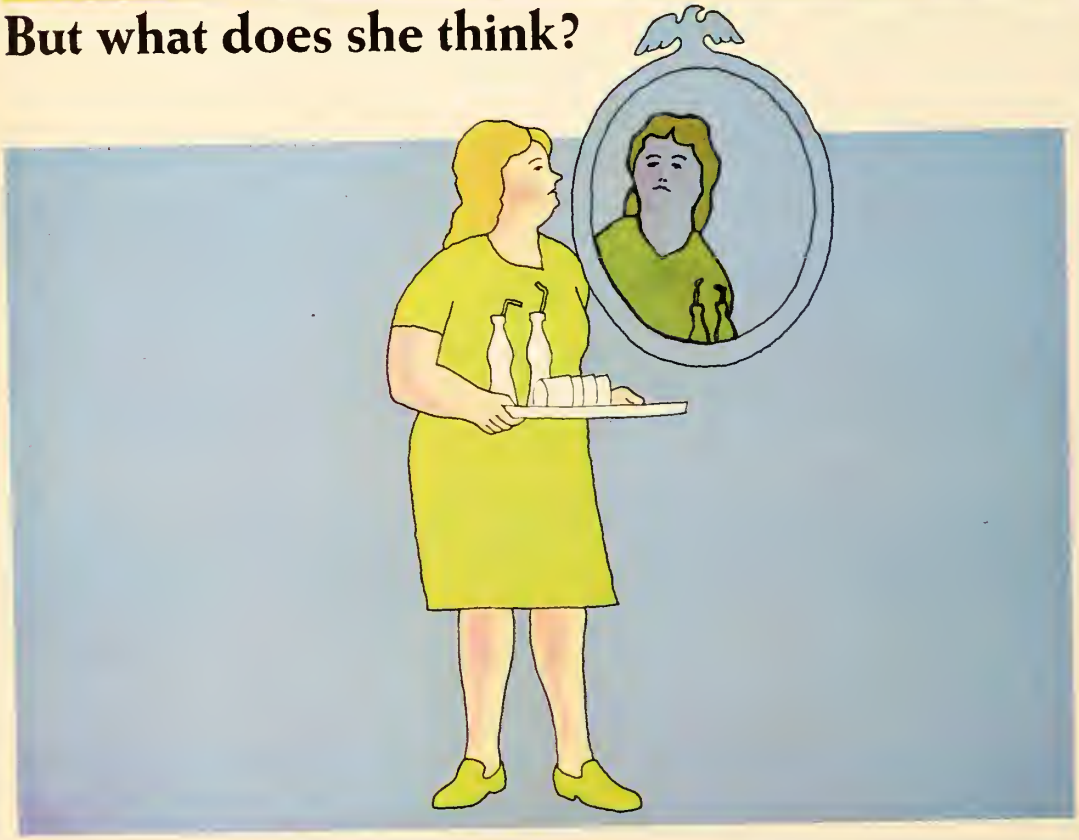
Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

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Dextro-amphetamine sulfate: Excessive use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring alertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libida, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulacytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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An Unusual Case of Gout

GEORGE J. ROBERTSON, M.D.

The distinctive clinical features of acute gouty arthritis leads to its ready differentiation.

Acute attacks involve at first one or a few joints. Exquisite pain, swelling, and tenderness are characteristic. The great toe (Podagra) is a favored site of attack. Complete subsidence of inflammation with rapid return of normal joint function is the rule. Slow development of tophi after multiple attacks may require years. Familial occurrence is the rule. The patient is usually a male. The incidence is 95% male.

The following case of severe atypical gout with signs of systemic disabling disease is unusual.

CASE REPORT

A 62-year-old housewife was admitted to the Sisters' Hospital, Waterville, Maine, on February 5, 1963.

Chief Complaint: Severe joint pains and exhaustion.

Present Illness: An episode of "arthritis" in her neck and shoulders occurred twelve years prior to admission. This subsided without aggressive therapy and she remained well until three years prior to admission when a red, hot, swollen, and tender toe joint developed. Fever accompanied this. This was not in her great toe joint but "in one of the others." The episode lasted two weeks acutely and then slowly subsided. Subsequent attacks occurred frequently and involved multiple joints. Complete subsidence between acute episodes was denied. A diagnosis of rheumatoid arthritis was tendered in 1961 in New Jersey. Aspirin "helped some". A trial with Colchicine was unsuccessful. Exacerbations occurred with emotional upsets, and spontaneously. The present episode began in November of 1962 with acute pain and swelling of the fifth finger of the left hand. It gradually subsided by Christmas leaving "a little white pearl".

By this time, other fingers were involved and by New Year's Day eight fingers, eight toes, both elbows, both knees were acutely involved, and she was bedridden because of exquisite pain on the slightest motion. Drenching night sweats occurred. Marked weakness rapidly developed. There were no chills. She did not check her temperature.

Medications taken during the three years of arthritis were Aspirin, Colchicine trial, three injections of Gold during the present episode, Panalba,® and Decagesic.® The latter seemed to be the most helpful in decreasing pain.

Past History: Usual childhood diseases. Good health until onset of arthritis. She admitted a cocktail or two before her evening meal. (Her son revealed her intake to be one-half to one pint of whiskey daily for several years.) Dietary history seemed adequate. She smoked one-half to one package of cigarettes daily.

Family History: No known gout or other arthritis. Tuberculosis in a grandparent.

Systems Review: Glaucoma controlled by drops. No cardiopulmonary, GI, or GU symptoms, or past disease. Menopause occurred in her forties.

Physical Examination: The physical examination revealed an acutely-ill lady with startling, swollen, red, hot, and fluctuant joints. The proximal interphalangeal joint of the left, fifth finger revealed a mountainous swelling, and an angry, red fluctuant mass with focal yellow spots. The distal interphalangeal joints of the third fingers and the proximal interphalangeal joints of the second fingers were similarly, but less, involved. There were yellow, pustular-like blisters of material on the pads of the left thumb and the lateral portion of the distal phalanx of the index finger. The distal interphalangeal joints of the second and third fingers also had pustular-appearing masses. Questioning revealed the yellow material had appeared only a few days before admission. The left wrist was thickened and tender. The posterior elbows had large (walnut), red, sagging, fluctuant masses with similar yellow content. There was a 45° flexion deformity of the right elbow. The knees were swollen. They were not red. There was a flexion deformity of 20° of the right knee, of 90° of the left knee. Flexion of the left foot was not possible. The dorsum was red and tender. Other joints were normal. There was marked generalized muscular wasting. The blood pressure was 130/80, pulse 120, temperature 99.6°, respirations 20. The lungs were clear. The breasts, female, revealed no masses. The abdomen revealed prominent lateral veins. The liver edge, smooth and definite, was felt two fingers breadths below the right costal margin. The spleen was not palpable. Pelvic and rectal exams at a later date revealed no abnormalities. Neurologic examination also done later revealed no abnormalities.

Admitting Diagnosis: 1. Gouty arthritis.
2. ? of cirrhosis of the liver.

Laboratory:	Hemoglobin:	10 grams
	Hematocrit:	31%
	Serum uric acid:	6.5; 5.7; 4.2; 8; 3.2
	24° urine uric acid:	Control – 640 mgms. ASA – 1260 mgms. Benemid® – 3200 mgms.
	Latex Fixation:	Negative.
	Lupus Erythematosis:	Negative.
	Bromo sulpho-phthalein:	3.5%
	White Blood Count:	8500
	NPN:	24 mgms.%
	FBS:	97 mgms.%
	TP:	5.8 grm. with A?G = 2.9/2.9
	Serum creatinine:	0.9 mgms.%
	Serum Iron:	64 micrograms%
	Stools x 3:	Negative for occult blood
	PBI:	3 micrograms %
	Pap Smear Cervix:	Negative.
	Pustular Material ⟶	Urate Crystals.
	x-ray	*Chest:* "Inactive fibronodular apical tuberculosis. *Hands:* "Cystic radiolucencies consistent with gout."

Hospital Course: One of the pustules at the end of a finger was aspirated and examined under the microscope on the night of admission and revealed a field of needle-like crystals. Colchicine, 0.5 mgms. q 1°, brought about nausea and vomiting with the 18th tablet. Her joints subsided remarkably. The massive inflammation of the left, fifth, finger collapsed, ruptured, and discharged an egg-shell-like, pasty material. Joint pain and swelling had completely subsided 48 hours after admission. On the 15th of February an upper-respiratory infection occurred. She was by then on Benemid and Colchicine, gms. 1 and 1.5 mgms. daily, respectively. An acute attack of arthritis occurred on February 17th. She refused a second intensive course of Colchicine. This attack subsided with Oxyphenyl Butazone. Her night sweats continued, were found to be head and upper thorax sweats, and were relieved with Diethylstil-

besterol 0.25 mgms. daily. Physical therapy brought about ambulation. Because of puffy-appearing lids and husky voice a PBI was done, found to be mildly depressed, and thyroid extract, gr. 1 daily, was added to her regimen. She refused all large tablets, claiming she never could swallow them. She was discharged to her own community hospital on Arthropan® (Choline salicylate) liquid, 4 i d, Thyroid gr. 1 q d, Diethylstilbesterol 0.25 mgms. daily.

Follow-up Office Visit: August 9, 1963. "She feels remarkably well. There has been one mild attack of gouty arthritis involving the mid-phalangeal joint of the third finger and the right foot.

Physical exam revealed the appearance of health. Muscle mass markedly increased since last seen. Joints supple with no restrictions. Nodules on both elbows felt firm and fleshy and could not be distinguished as tophi."

Laboratory:	Serum uric acid:	6.0 mgms.%
	Hemoglobin:	12.5 grams
	Hematocrit:	36%
	Sedimentation rate:	38% (Wintrobe)
	White blood count:	8100
		P-66, L-30, M-2, E-2.
Urine:	pH	5.0
	Specific gravity:	1.009
	Albumin:	Negative.
	Sugar:	Negative.
	Sediment:	1-2 wbc/hpf
		Negative casts.
	Biopsy of left elbow nodule ⟶	urate tophus.

Discussion

The readily available tophi lead to prompt diagnosis. The response to Colchicine was classic. Further history on her previous trial with Colchicine revealed that the drug was discontinued after 5-6 tablets because of subjective nausea. The importance of adequate trial with Colchicine is evident.

The first rheumatic attack twelve years ago seems unrelated to gout. It is assumed that her clinical gout was of three years' duration and was of unexpected severity. Indeed, the pattern of her disease prior to the appearance of tophi suggested the diagnosis of rheumatoid arthritis. The muscular wasting, hypoalbuminemia, anemia, are not described in the current literature on gout. The complete response to Colchicine, the biopsy of the elbow nodule, the negative latex fixation test for R.A. are all evidence that she did not have rheumatoid arthritis. The above elements suggesting systemic disease can readily be explained by sustained inflammatory joint disease in this lady. It is difficult to find well documented cases of rheumatoid arthritis and clinical gout in the same patient. Two-hundred and six case records of clinical gout were reviewed by this discussor at the New England Center Hospital. None had rheumatoid arthritis. Five with severe tophaceous gout had muscular wasting, anemia, hypoalbuminemia. These have been reported by Gowans.[1] All responded to adequate acute and interval therapy and the signs of systemic disease cleared as they did in this patient. Hutchinson[2] in 1881 described the concurrence of the two diseases. Talbot,[3] in his latest text on gout, mentions a patient in his series with tophaceous gout and histologic evidence of rheumatoid arthritis. The combination is apparently rare and perhaps rarer than one might expect from chance coincidence in two common diseases.

One might speculate on the reason for unusually severe and progressive gout over three years time in this female. Gout appeared at the proper time, i.e. postmenopausal in this lady. Family history was unreliable. Familial gout is assumed in the absence of pharmacologic agents and diseases associated with secondary gout. Hypothyroidism was probably present in this patient although good documentation was not obtained. Leeper[4] has reported higher mean levels of uric acid in myxedematous males and females compared with controls. The elevation was attributed to decreased blood flow. Significant contribution by this factor seems unlikely in this patient.

Alcohol in some patients seems to be a factor in precipitating attacks. Since this patient drank steadily and moderately to heavily, could this account for the severity of her disease? Lieber et al[5] observed increased concentrations of uric acid in patients intoxicated with alcohol. It was suggested that changes in renal transport of urates were produced by the increase in blood lactate associated with oxidation of ethanol. Seegmiller et al[6] also notes that alcohol may be a provocative agent in some patients and that alcohol increases the serum urate level through the urate-retaining action of lactate to which it gives rise. Alcohol may indeed have been an attack-provoking agent in this patient and perhaps through the same mechanism a means of prolonging the attacks. Her present attack-free course may in part be due to restriction of alcohol.

Of some interest are the levels of serum uric acids obtained in this patient. Fig. 1 is a graph of the patient's hospital course. The dotted line represents uric acid levels in serum. The peaks in the smooth line represent the two attacks of gout. The highest serum uric acid reported in this patient was 8 mgms. %. Several determinations had been done in various places according to her referring physician, and the 6.5 mgms. % in January of 1963 was the highest seen and led to the trial with Colchicine. She had severe acute tophaceous gout on admission. Her serum uric acid on the morning after admission was 5.7 mgms. %. All uric acid determinations were by colorimetric method utilizing the ability of urate to reduce phosphotungstic acid to a colored product. The errors in this method are recognized, but enzymatic spectrophotometric methods were not available to us. The clinically important points to be gathered from this chart are:

1.) that several uric acid determinations may be necessary in a suspected case of gout. Seegmiller et al[6] report 9% of gouty patients may have serum uric acids below the upper limits of normal at times.

2.) that tophaceous gout can be seen with normal levels of serum uric acid. Seegmiller et al[6] state that in their experience they have seldom seen tophaceous gout

CLINICAL COURSE

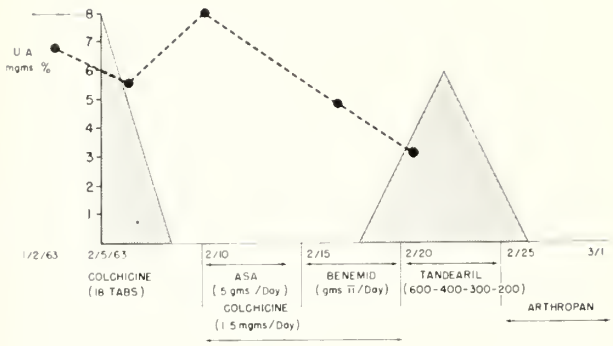


Fig. 1

until the serum urate rises to values consistently above 8 mgms %.

3.) that acute attacks can occur in the presence of normal serum uric acids. The attack of acute gout noted in Fig. 1 started four days after Benemid and two days after the onset of an upper respiratory infection. This attack, therefore, could be a so-called Benemid attack or an attack produced by the stress of the URI. The mechanism of attacks of gout following the onset of therapy with uricosuric drugs is not understood and seems to refute the hypothesis that Seegmiller[6] has proposed for the pathogenesis of the acute attack (Fig. 2). He has proposed that in such attacks as these, the urate crystals are liberated from resolving tophaceous deposits rather than from hyperuricemic body fluids.

4.) that Colchicine is not a uricosuric agent. In spite of the dramatic relief of arthritis obtained with Colchicine, the uric acid level reached its highest recorded level after Colchicine and before uricosuric agents were added to the therapy.

The determination of quantitative urinary uric acids is an integral part of a work-up in the patient with gout. On a low-purine diet this patient had a 24° control urinary uric acid of 640 mgms. % (normal 200-600 mgms. %). Aspirin, 5 gms., daily, had a definite uricosuric effect – 1260 mgms.

It is not possible to state from these meagre studies whether this patient was a normal or a hyperexcreter of uric acid. Both the serum uric acids and the urinary uric acids may have been influenced by the unknown amount of medications, such as steroid and salicylates, which she received prior to admission. In most cases, however, a daily excretion of uric acid in the urine of 600 mgms. or over, while on a low-purine diet and without uricosuric medication, is an indication of overproduction of uric acid in the body.

Gutman[7] states that urate-stone incidence is about 40% in over excreters of uric acid in primary and secondary gout, and 20% in normal excreters of uric acid in primary gout as compared to 0.01% in non-gouty patients. If urinary calculi can be prevented by any means, then it would seem worthwhile to attempt such in patients with large amounts of uric acid in their

SUGGESTED MECHANISM OF ACUTE ATTACK

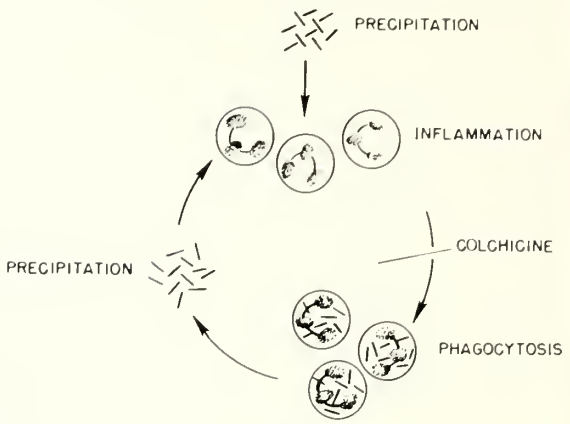


Fig. 2

Solubility of Uric Acid in Buffered Solutions

	Uric Acid (mgms/100cc)
Serum, pH 7.4	7
Urine, pH 5.0	15
Urine, pH 7.0	200

Fig. 3

urine. Two measures are available. The first is to maintain a high fluid intake. The second is suggested by the solubility of uric acid in urine at varied pHs. Fig. 3 taken from Seegmiller et al[5] reveals a marked increase in uric acid solubility in alkaline urine. Our patient has no history of stone and at present is on a high-fluid intake, 3000 cc. daily. Should stones occur, alkalinization with sodium bicarbonate and Diamox® utilizing pH paper for the patient testing of urine will be instituted.

SUMMARY

A patient with severe tophaceous gout accompanied by signs of systemic disease is presented. Although anemia, hypoalbuminemia, and marked muscular wasting accompanying gouty arthritis are not mentioned in current literature, a review of 206 cases of gout admitted to a University hospital revealed five similar cases.[1] These patients respond to conventional therapy for gout with disappearance of the muscular wasting, anemia, and presumably the hypoalbuminemia. Rheumatoid arthritis was not present in this patient. Gout and rheumatoid arthritis apparently occur rarely in the same patients. The possible relationship of the associated disorders, hypothyroidism, and alcoholism, to the frequency and severity of this patient's gout is discussed with no conclusions being possible. The serum uric acids in this patient were not as high as one might expect in tophaceous gout. The importance of quantitative urinary uric acid is stressed, especially in relationship to possible means of decreasing the frequency of urate calculi.

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Series Defect in School Failures with Adequate I. Q.'s

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In the latter half of the 19th Century, some beginning investigations in the problems of learning were undertaken. Wertheimer headed some work on serial learning that was responsible for the development of Gestalt Psychology. Very shortly, the studies in serial learning were eclipsed by the greater importance of the Gestalt Psychology, and almost forgotten. A linkage was made by Javal and others, between neurological signs and learning. These studies were also eclipsed by the spectacular advances of Freudian Psychiatry. In the 1930's, Orton advanced the theory that crossed dominance was responsible for learning problems. At the same time Sperry, Strachey and Rubenstein stated that environmental influences were considered to be practically the only cause of learning defects. In the 1940's, Kramer and Pollnow demonstrated that overactivity and short attention span were associated with brain damage in children. Bender and Goldstein developed tests for brain damage in which the patient is asked to reproduce geometrical designs.

Recently, there has been a rebirth of interest in the causative nature of physical defects in learning problems. Wagenheim has shown a definite statistical relationship between learning problems and a child having contracted one of the childhood diseases before the third birthday. De Hirsch has stressed the need and demonstrated methods of discovering those children who will become school failures at a preschool level. In 1964, Birch and Belmont tested children's temporal order by having them match Morse Code signals with the printed dots and dashes.

Children with learning problems sometimes masquerade as mentally retarded or emotionally disturbed youngsters because learning problems in children are related to the way language is handled in the brain. In some cases, true aphasia may exist where definite organic brain damage has occurred. In many children no record of injury can be found, but it is quite possible that an improper development of the brain may exist. Evidence has been accumulating that heredity influences many forms of behavior, including learning problems. Anoxia at birth in the form of delayed respiration, has been found to slow the social development of children for the first two years of life. Differences in social background and the presence of parental anxiety have also been cited as causes of slow or abnormal development. Some investigators have found changes in EEG's in psychotic children, and that overactive patients have different EEG's than normal persons. Thus, overactivity is often associated with learning problems.

Studies have shown that children who have a reading disability appear to overcome these handicaps with time, and have a tendency to show few residuals of their defects as adults, other than those of a poor educational background. A period of several years is necessary to demonstrate school failure to a point where it is noticeable in children. Usually, unmistakable signs have appeared by the time the child reaches third grade. The investigator's feeling is that the third grade represents a time in a child's development that is most likely to highlight definite statistical differences between children with learning problems and those without such problems.

The present investigators have found two areas of behavior that seem to show specific lacks in children with learning problems. One, is in the area of serial learning and has been called series defect.

MATERIAL

Over the past six years, we have been aware that children who had school problems were apt to find it difficult to repeat things in series. As a result of this observation, one of the authors decided to test all children under his care who were having school failures, to see how many of them were actually having difficulty with series. During a three-year period, eighty children were thus studied from three settings; private practice, the Portland Child Guidance Clinic, and the Sweetser Children's Home. Of these, forty-seven were found to have a series defect, while the remaining thirty-three had not.

A series defect is considered to be present if a child is unable to report things arranged in series, which children of comparative age are usually able to do successfully. In this study, children were asked to count from one to twenty. Most children are able to do this by the time they have completed sub-primary, or by the time they have attained a mental age of about six years. The children were then asked to repeat the days of the week in order, an item at Year VIII level on the Stanford-Binet Intelligence Scale. They were also asked to say the alphabet, which most children are able to do at the beginning of the third grade, or at about the age of eight. Finally, they were asked to give the months of the year in order, a task which is usually learned at the age of ten. Any failure to perform these tasks at the expected age level was considered to be a positive sign of series defect.

To check the usual ages at which children without school problems were able to do these tasks successfully,

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the first fifty children who were seen at the Portland Child Guidance Clinic after Jan. 1, 1963, who were six or more years of age with an IQ of 85 or above, and were not experiencing any difficulty in school, were given the above tests. It was found that no child six or more years of age was unable to count to twenty. No child seven or more years of age was unable to repeat the days of the week in order. Only one child at age eight failed to say the alphabet correctly, and all children over the age of ten were able to give the months of the year in order. In this group of fifty, there were: four children aged 6; four, aged 7; seven, aged 8; four, aged 9; four, aged 10; twenty-seven, aged eleven or older.

For the purpose of this study, a school failure was defined as a child with any two of the following symptoms at the time of initial evaluation.

1. Failure in one subject, or a rank of D in two subjects.

2. Achievement, as indicated by the Wide Range Achievement Test, in Reading, Spelling or Arithmetic, of at least one grade behind school placement.

3. Repetition of at least one grade in his school career.

Children who had displayed none of these difficulties were considered free from school failures.

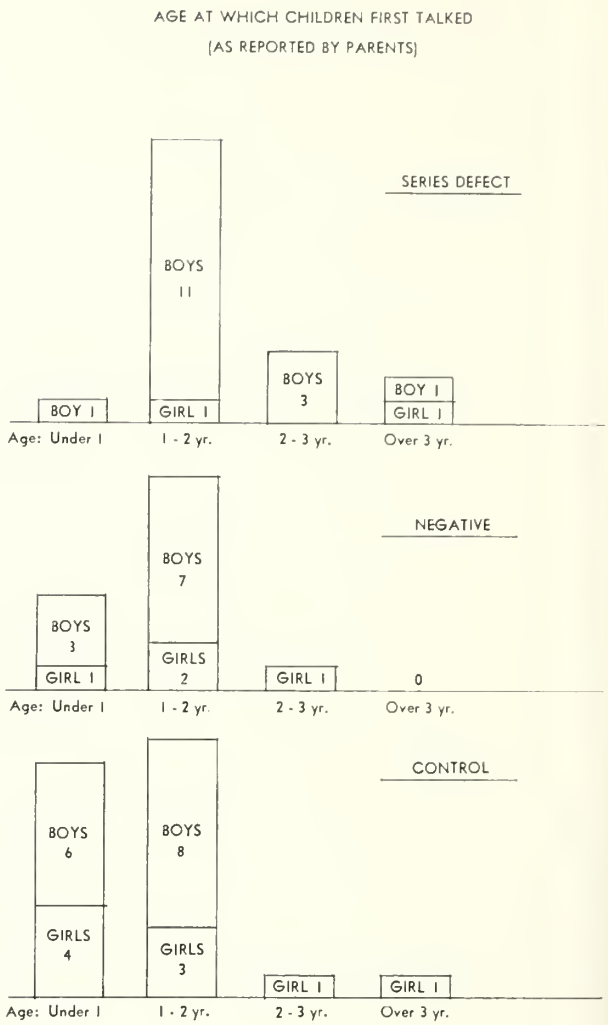
All the children in this study were over the age of eight, and had IQ's of 85 or above. As was mentioned before, eighty children with school failures were studied. Of these, forty-seven had series defect. (Series defect seemed to become less intense with increase in age.) Thirty-three were found not to have series defect, and make up the so-called Negative group. A group of fifty children who had been seen at the Portland Child Guidance Clinic between Jan. 1, 1961 and Jan. 1, 1964, who had no signs of school failure, were eight or more years of age, and whose IQ was 85 or over, were selected by starting alphabetically and proceeding until fifty such children were found. This group was designated as the Control group. This type of Control group was used because all so-called normal groups of children contain a high percentage of disturbed children and are much more heterogeneous than the Control group.

We have divided our studies into three groups: A. Those studies in which statistically significant findings are shown; B. Those studies in which statistics suggest no real differences exist; C. Those studies in which trends are present to a non-significant degree.

A. *Statistically Significant Findings* – Children with series defect are almost entirely boys, whereas, approximately two-thirds of the Negative group are boys, and only slightly more than half of the Control group are boys.

Sex of Children with Series Defect
As Compared to Negative and Control Groups

	Boys	*Girls*		X^2
Series Defect	44	3	Series Defect vs. Control	17.9
Negative Group	23	10	Series Defect vs. Negative	8.2
Control Group	28	22	Negative vs. Control	1.5



We asked ourselves several questions concerning the children with series defects. A question that seems important is – Are the children in the Series Defect group more, or less, disturbed than children in the Negative and Control groups? In order to determine this, we turned to the case histories of the children involved and selected items from them that might be expected to be found in children who were emotionally upset, and compared their incidence within the three groups. Fortunately, most of the case histories had been taken by the same psychiatrist, or by persons trained by him, so there was a uniformity as to the inclusion of the desired material in the initial interviews. We had previously found that disturbed children were more apt to say that their daydreams seemed real to them, to complain of frequent fatigue, to have more food fads, see less of their friends, like to be by themselves more, tire more easily doing nothing, than do normal children. It was found that children in the Control group were more apt to have these symptoms than the children with series defect. It was also shown that the Negative group might show divergences in the same direction as the Series Defect group, but not to a significant degree.

		Daydreams Seem Real	*Has Food Fads*	*Sees at Least 1 Friend Out of School Within the Week*	*Likes to Be With People*	*Tired Doing Nothing*
Series	Y	3	21	34	36	8
Defect	N	30	23	10	8	36
Negative	Y	7	21	20	22	9
	N	26	10	10	11	24
Control	Y	12	31	24	20	20
	N	27	11	21	18	18
X^2Con.-Pos.		5.1	5.6	6.2	8.0	10.8
Con.-Neg.		<1	<1	1.3	<1	4.7

It has been found that children who have difficulty with achievement in school are more apt to have trouble with speech early in life despite adequate intelligence, than those who do not have trouble in school. Children who have school problems also seem to have been slower in learning to speak. The time at which children first talked was compared in the three groups and children who had series defect talked considerably later than did those of the Control group. The difference was more than six times the probable error. The Negative group also talked later than the children in the Control group. (Graph, page 244).

As an afterthought, we felt it would be of interest to obtain some idea concerning the possibility of there being some relationship between series defect and some hereditary factor. We had no adequate figures with which to do this. People who have trouble in school would be less likely to graduate from High School than those who had experienced no difficulties. Therefore, we felt that we could get some idea concerning this by telephoning the parents in all three groups and asking them who, in their family, graduated from High School. We did this by asking each parent whom we were able to locate, about the child's grandparents, parents, aunts and uncles. There were some interesting sidelights which showed up. The people in the Control group had a tendency to have moved or disappeared from the community to a much greater degree than people whose children were in the Series Defect or Negative groups. Many of them were also quite belligerent in their responses; some even refused to give the desired information. None of the parents in the Series Defect or Negative groups refused to cooperate.

When we compare the number of family members who did not finish High School with those who did, in the three groups, we get the following statistics:

It was found that the fathers of the Series Defect group were more likely not to graduate from High School than were the fathers of the Control or the Negative groups. The maternal grandfathers of the Series Defect group were more likely not to graduate than those in the Control group. Maternal uncles in the Negative group were more apt not to graduate from High School than those in the Control group. These findings, even though based on rather inaccurate measures for the presence of hereditary factors, would suggest that there is some connection between hereditary factors and school failures in families whose children have series defects.

B. *Those studies in which statistics suggest that no real differences exist* – In the past, a great deal has been said about crossed dominance having a serious effect on school failures, more particularly in persons with Reading and Spelling defects. We found no significant differences between the three groups.

Percentage Of Children Who Showed Crossed Dominance In The Groups

Control 17.5% Series Defect 17.5% Negative 23%

It has also been claimed that children who have a non-existent father were more apt to have school failure than those who had adequate fathers. The number of children whose father was dead, severely ill, an alcoholic, a non-provider, employed away from home, who was passive or sociopathic, whose parents were divorced or separated, and those children who were placed out of the home, was compared to the number of children who had relatively normal relationships with their fathers. There were essentially no differences between the groups.

Percentage Of Inadequate Fathers Found In The Groups

Control 56% Series Defect 60% Negative 62%

C. *Those studies in which trends are present, to an insignificant degree* – Wagenheim suggests that boys who contract measles, German measles, chickenpox or mumps before the age of three, are apt to be troubled by school failures. It was found that boys in the Negative group showed a tendency to be more apt to have had these illnesses and whooping cough, before the age of three, than were those in the Control group. Actually, the figures in the Negative group are not conclusive, but show a tendency to fit with Dr. Wagenheim's findings.

		F	M	MGF	MGM	PGF	PGM	Mat. Aunts	Mat. Uncles	Pat. Aunts	Pat. Uncles
Series	Passed	5	7	5	6	4	5	13	11	22	14
Defect	Failed	12	10	12	11	9	9	7	5	6	7
Neg.	Passed	14	18	10	15	10	10	18	26	10	21
	Failed	10	6	14	8	14	14	5	3	6	6
Con.	Passed	12	12	13	11	9	8	6	11	11	21
	Failed	7	7	6	8	10	11	0	5	8	7
X^2 Pos.-Con.		3.2	2.1	5.4	1.8	<1	<1	<1	0	2.4	<1
Neg.-Con.		<1	<1	<1	<1	<1	<1	1.5	3.1	<1	<1

	Series Defect		Negative		Control	
	Illness before 3	*No illness*	*Illness before 3*	*No illness*	*Illness before 3*	*No illness*
Boys	8	18	10	9	5	10
Girls	0	3	5	5	10	11

X^2—Neg. vs. Con. 1.3

There were no children who were diagnosed as schizophrenic in the Series Defect group. Two were so diagnosed in the Negative group and six were found to have schizophrenia among the Controls. Even though these figures are not significant, does this mean that schizophrenia is less apt to be found in series defect children than in the usual population of a Clinic? If this is so, only further study of a larger number of cases would be of help in determining the answer to the question.

Discussion of Treatment

Most children who have serious problems with school failure have emotional difficulties as well, and require psychotherapy. However, the most important part of the treatment is remedial education. This proves to be quite effective if it is associated with psychotherapy.

Remedial education should, first of all, be started at the achievement level at which the child is presently functioning. Many untrained tutors start the child at his grade placement and a complete failure results. Secondly, since this appears to be an aphasic type difficulty, every sensory and motor modality should be used in an effort to allow the child to find neural pathways that will circumvent the probable cortical lesion. From a practical point of view, this may often be done by using games, such as; word bingo, fish, with word cards which have related sounds, word and picture dominoes, etc. Form is also very important in teaching these children, three-dimensional concepts such as letters and three-dimensional models, as the feel of these objects is most helpful. Both models and pictures are helpful in the games described above. Work with speech problems is also of utmost importance.

Case Report

Age 14, second of five children, the patient characterized his siblings as "big tease, tries to run me and mother, upsets me, spoiled rotten, always very unpleasant." He also said "everything I try to do causes a fight between mother and father."

He repeated first and fourth grades and when first seen, was in the seventh grade. During the year, he had had trouble in school three times, for which he had to see the Principal. The Principal felt the child was not at fault.

He did not have many friends and only saw them at school and at Scouts. He felt that people talked about him and that he was an odd ball, "smart but dumb." He felt that people would rather leave him than take him. He believed that he had control of his pores and that he could make a certain portion of his body sweat while wrestling, so that he could slip away from his opponent. He was unable to learn to read.

He felt that he would do better studying if he could study some place other than home. He despised his father for telling him he wasn't worth the powder to blow him to hell. He was afraid of his father, but respected him for being strict.

Psychological tests: October, 1961. WISC – Verbal 116, Performance 106, Full Scale 112. Wide Range Achievement Test – Spelling 1.9, Reading 1.9, Arithmetic 3.6. A severely disturbed Rorschach picture was noted. He was unable to count to 20 correctly. He could not repeat the days of the week or months of the year in order correctly, and was unable to say the alphabet.

The patient was given individual and group psychotherapy. Remedial education was offered as was therapy for his parents. His parents came in only a few times and did not feel they could afford remedial education at this time.

During psychotherapy, familial relationships of an oedipal nature were discussed. He was encouraged to enter into social activities and did join a Judo class where he had the first really successful experience of his life. He became more friendly with his peers. His family relations cleared to some extent and he lost most of his feeling of depersonalization and difference.

In June, 1962, he had a reading grade of 2.2, Spelling 2.1 and Arithmetic, a grade of 3.5 on the Wide Range Achievement Test.

During the summer he continued therapy until he entered the Sweetser Children's Home in September of 1962, at which time remedial education was begun, and psychotherapy was continued on a family group basis, with individual therapy for the patient. It soon became evident that the father was a logical individual who was incapable of insight. This man soon found excuses to discontinue treatment, although the mother did attend regularly until family therapy was discontinued by the Institution in November of 1963. On admission to Sweetser he had a Reading grade on the Metropolitan of 1.6, Spelling 1.8 and Arithmetic 3.8.

The patient never showed the relatively severe emotional disturbance at Sweetser that had been present when he was first seen by the Clinic, although he was afraid of the dentist to a point where it was necessary to give him a general anesthetic in order to work on his teeth.

By the following June, 1963, he was doing fifth grade work in Reading, in Spelling he was about half way through the third grade level, and he was doing sixth grade work in Arithmetic.

In the fall of 1964 he entered ninth grade in a local, public, Junior High School. He was placed in the third track, (a group of poor achievers, all boys). His grades at the end of the first ranking period were: English C (83), Math C (80), Civics C (78), Industrial Arts B (86), in which class he was reported to be the best worker in the class, Home Management B (87). He is getting along well with everyone at school and is not a disciplinary problem.

Discussion

This paper is a preliminary report of our findings on those children who have a series defect. It seems that these children are not a homogeneous group. For one thing, the severity of the symptom varies with the age of the child, and from child to child. In some, it appears to be simply one of many signs of brain damage. In others, it appears to be the central sign around which many symptoms are built, as in the case reported here.

There are many areas that would be interesting to explore further in relation to series defect. It would be interesting to know what correlation existed between series defect and psychological tests for brain damage such as the Bender-Gestalt, Hooper Visual Organization Test, etc. It would also be of interest to study the relationship between series defect and the presence of definite emotional illness. Is there a correlation between series defect and other neurological signs often found in

children, such as stabismus, cerebral palsy etc.? A large number of sample cases would also be of help to clear up whether some areas that have been reported as showing tendencies of being related to series defect were actually related, or not.

We do feel that series defect is a definite sign of brain damage found in children showing school failures. There is a strong suggestion that series defect, in some cases, is a sign of hereditary difficulty.

Summary

1. Series defect is found in over half the children having problems with school failure.

2. School children with series defect are mostly boys.

3. Children with series defect learn to talk later than most children.

4. The incidence of series defect appears to have no relation to having childhood diseases before the age of three.

5. Having had childhood diseases before the age of three is found to be important in those children who have school failures without series defect.

6. There appears to be no relationship between crossed dominance and school failure.

7. The evidence of a nonexisting or weak father figure seems to have little relation to school failure.

8. Series defect may be related to hereditary factors.

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AN UNUSUAL CASE OF GOUT — *Continued from Page 242*

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171 Harrison Avenue, Boston, Massachusetts

Behind continued high blood pressure readings lies the possibility of organic damage[1-13]

MANY OF THE aspects of essential hypertension are unpredictable—either because there are a number of mechanisms involved or because individuals differ in their responses to these mechanisms.[1]

There is one aspect of hypertension, however, that seems, in many cases, predictable. ". . . when the blood pressure is elevated to a marked degree for an adequate period of time, this in itself leads to perpetuation of the syndrome with resulting vascular damage throughout the body."[14] All too often the disease progresses until there is damage to one of three vital organs: the heart, the kidney, the brain.



"Hypertension is certainly a major factor in the genesis of coronary heart disease, and it is even more important when compounded with obesity."[4]

"[Vascular deterioration] can be clearly seen in the kidney with a degree of damage that can be measured by renal function studies."[10]

". . . most evidence suggests that reduction of blood pressure, when it is too high, not only relieves the heart of excess work but reduces vascular damage."[1]

"In short, treatment is indicated."[1]

Antihypertensive therapy will not restore the blood vessels to normal. Yet many of the vascular changes and symptoms caused by increased blood pressure may be arrested or alleviated when the blood pressure is reduced to normotensive levels.[7]

Reducing the blood pressure helps curtail further vascular damage and improves the prognosis — when damage is not too far advanced before therapy is started.[14] Essential hypertension is an indication not only for treatment, but for *early* and adequate treatment of the patient in question.

Reduce the blood pressure with Rautrax-N

Rautrax-N combines the antihypertensive-tranquilizing action of whole root rauwolfia with the antihypertensive-diuretic action of bendroflumethiazide in one convenient medication. The two drugs complement each other so that smaller doses of both are possible.

Rauwolfia combined with bendroflumethiazide is particularly effective in long-term therapy,[15-17] since beneficial effects do not diminish with continuous daily administration.

For most patients 1 or 2 Rautrax-N tablets daily are sufficient for maintenance therapy. The simplicity, convenience and economy of such a dosage schedule are of particular benefit to older patients.

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Contraindications: Severe renal impairment or previous hypersensitivity. **Warning:** Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting or G.I. bleeding occur.

Precautions and Side Effects: The dose of ganglionic blocking agents, veratrum or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Caution is indicated in patients with depression, suicidal tendencies, peptic ulcer; electrolyte disturbances are possible in cirrhotic or digitalized patients. Marked hypotension during surgery is possible; consider discontinuing two weeks prior to elective surgery and observe patients closely during emergency surgery. Rauwolfia preparations may cause reversible extrapyramidal symptoms and emotional depression, diarrhea, weight gain, edema, drowsiness may occur. Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients, and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, rashes may occur.

Dosage and Supply: Initial dosage, 1 to 4 tablets daily, preferably at mealtime. Maintenance, 1 or 2 tablets daily. Rautrax-N is supplied as capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin®), 4 mg. bendroflumethiazide (Naturetin®), 400 mg. potassium chloride.

Also available: Rautrax-N Modified — capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin), 2 mg. bendroflumethiazide (Naturetin), 400 mg. potassium chloride. Both potencies available in bottles of 100. For full information, see Product Brief.

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Squibb Rauwolfia Serpentina Whole Root (50 mg.) with Bendroflumethiazide (4 mg.) and Potassium Chloride (400 mg.)

SQUIBB 'The Priceless Ingredient' of every product is the honor and integrity of its maker.

DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Maine's Expanded Medical Care Program — Title XIX

There has been much discussion and some confusion during the several weeks which have elapsed since July 1, 1966 when a considerably broadened medical care program was implemented by the Maine Department of Health and Welfare on behalf of its public assistance recipients.

This program is based on PL 89-97 legislation, of which Title XIX is a part.

Initiated in this issue, and to be continued next month is a compilation of communications with physicians, hospitals, municipal officials, home health agencies, other suppliers of medical equipment or services. This will provide physicians with comprehensive information concerning the Department's Title XIX medical care program, and, hopefully, serve as a useful reference. In this issue, the communications from Dean Fisher, M.D., Commissioner, Maine Department of Health and Welfare to physicians licensed to practice in Maine are reproduced. They reflect current procedures.

June 29, 1966

SUBJECT MEDICARE — TITLE XVIII, PART B — MEDICAL CARE FOR PUBLIC ASSISTANCE PATIENTS AGE 65 AND OVER

Starting July 1, 1966 essentially all Public Assistance Recipients age 65 and older (Old Age Assistance) will be covered under PL 89-97 – Title XVIII, Part B in a similar manner as are Social Security beneficiaries in this age group. However, in addition to the medical care services available under Title XVIII, the Department will also pay for the following additional services:

1. necessary routine physical examinations
2. immunizations
3. all outpatient services
4. hospitalization beyond 90 days for each "spell of illness" if necessary
5. home health services beyond 200 visits if medically necessary
6. first three pints of blood

Please treat and then bill for these patients through the fiscal intermediary (Union Mutual Life Insurance Company) as you would any other patient under Part B.

For these patients you will bill for 100% of your reasonable charge rather than the 80% applicable to the Social Security beneficiary. You are urged to bill the fiscal intermediary directly instead of the patient as this will allow for faster processing of claims and faster payment to you.

A letter has been mailed to each Old Age Assistance recipient asking that they inform you that they are receiving public assistance so that you will know when to bill for full payment.

June 29, 1966

SUBJECT "MEDICARE" — TITLE XIX — MEDICAL CARE FOR PUBLIC ASSISTANCE PATIENTS UNDER AGE 65

Starting July 1, 1966, the Maine Department of Health and Welfare will initiate a program under Title XIX of PL 89-97 for the financial support of medical services for public assistance recipients under the age of 65. This will include all recipients in the following categories:

1. Aid to Families with Dependent Children
2. Aid to the Blind
3. Aid to the Disabled

These recipients will be financed basically for the same scope of medical services which will be given under Title XVIII for those age 65 and over, plus the following additional services:

1. necessary routine physical examinations
2. immunizations
3. all outpatient services
4. hospitalization beyond 90 days for each "spell of illness" if necessary
5. home health services beyond 200 visits if medically necessary
6. first three pints of blood

For these patients the Maine Department of Health and Welfare will act as the fiscal intermediary and all bills should be sent directly to this Department. Please send bills to: Department of Health and Welfare, State House, Augusta, Maine with the required information on the enclosed forms which are similar to those to be used by the fiscal intermediaries for Title XVIII (Union Mutual Life Insurance Company). An initial supply is enclosed, others will be made available in the near future.

Fees will be paid on the basis of reasonable charges

similar to the payments which will be made under Title XVIII, Part B.

Recipients will be identified initially by a letter and later by an identification card which they will carry with them. This identification card cannot, however, be considered a final commitment by the Department because of the unpredictable factors in public assistance such as discontinuance or renewal of grants. We will, however, make every attempt to notify you immediately upon receipt of your report and bill if the individual is not currently covered by our services.

In order to complete payment to you as quickly as possible, it is hoped that you will bill the Department rather than the patient. Our principle effort will be to make the procedure of financing medical care for our recipients such that there will be a minimum of paper work for you and for us.

August 26, 1966

SUBJECT PAYMENTS FOR MEDICAL CARE FOR PUBLIC ASSISTANCE RECIPIENTS

We have recently obtained an additional interpretation of the Title XIX medical care legislation. This interpretation deals specifically with the "vendor" payment aspect of Title XIX, PL 89-97. As we previously indicated to you, Title XIX regulations provide only for a "vendor" payment which is a payment directly to the person or agency providing care. This simply means that neither a public assistance recipient nor other individual or municipality can be reimbursed for payment made to a physician or other provider of service. There is no provision in Title XIX for "assignment" or non-assignment of benefits.

The Department of Health and Welfare is paying the $3.00 monthly premium, the $50.00 deductible and the 20% co-insurance charge associated with Title XVIII, Part B, in behalf of all Old Age Assistance recipients. These expenses are paid from Title XIX funds and, thus are subject to Title XIX regulations. Therefore, the Old Age Assistance recipient who pays the physician for his services, cannot be reimbursed by the State through the fiscal intermediary for any part of the $50.00 deductible or 20% co-insurance charge. To reimburse would result in loss of Federal matching money.

We are aware that the new medical care legislation is complicated and we are equally aware that our previous communications with you have been written from our initial understanding of how Medicare (Title XVIII and Title XIX) would work. To assist us, and your public assistance patients, we are therefore, now requesting you to please: (1) direct all billing to the fiscal intermediary when covered medical services are provided to public assistance recipients age 65 and over; (2) direct all billing to this Department for covered medical care services provided to public assistance recipients under the age of 65.

After September 1, 1966, we can accept no receipted bill for reimbursement. We can only accept request for payment from physicians or other providers of service after that date via the procedures already established.

Furthermore, in order for the Department to authorize payment for physician's services, it is necessary to have in our files your signed statement of compliance with the Civil Rights Act. If you have not returned your individual signed statement, we would appreciate your doing so as soon as possible. After September 1, 1966, it will be impossible for this Department to make payments to any provider of medical services unless such a statement is on file in this office. Compliance with the Civil Rights Act is a mandatory aspect of all of our Federal matching programs, including Title XIX, even though this is not the case with Title XVIII. If you need another copy of the Civil Rights form, please let us know.

What Price Quality in Drugs?

We all know that it is illogical to expect a researeh-oriented eompany, that eommits itself to the diseovery of drugs that are needed as well as to the making of those that are popular, to meet the priees of the firm that doesn't. It is a mistake to expeet the firm that employs two quality eontrol teehnicians for every five employees to match the economic trieks of the eompany that does not.

—C. Joseph Stetler to annual Mountaintop Medieal Assembly, Waynesville, North Carolina, June 18, 1966.

AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

How Several Kinds of Hormones Work

Doctors at the University of California Medical Center, San Francisco, California have advanced a theory as to how several different kinds of hormones work. They say the hormones serve as a switch which turns on the gene machine.

On the basis of their experimental results, they postulate that the hormones act directly on DNA (deoxyribonucleic acid), the stuff that genes are made of. If this theory stands the test of time, it will supply one of the long-sought answers as to how animals and humans grow, develop highly specialized tissues and talents, and die.

This was reported recently by the American Cancer Society which has helped support the research done in the Cancer Research Institute by Dr. Melvin L. Goldberg, a senior medical student, and Dr. William A. Atchley.

The investigators worked with DNA extracted from human placental cells.

The DNA from these cells was incubated with each of several different hormones — including the female hormones, estrone and estradiol; the stress hormone, cortisol; sugar-burning insulin; growth hormone; and the heart and vessel stimulant, epinephrine. All these hormones (but not some others which also were tested) had the same effect; they weakened the bonds which tied the two strands of the DNA molecule together.

DNA is built like a zipper, in a way. It is composed essentially of two long segmented strands, which are joined together by hydrogen rungs. Each segment is made up of one of four bases and a phosphate group. Opposite segments complement each other as male and female, right and left hand, lock and key.

As the California scientists see it, DNA, when idle, is zipped up tight. When an effective hormone is introduced, it loosens the hydrogen cross pieces and unzips the molecule so that it is free to work.

The above situation was observed when the active hormones were applied to healthy DNA. It also prevailed when hormones were added to a normal DNA-and-protein combination such as exists naturally in chromosomes.

The hormones had no effect, however, when the DNA was damaged in some way, or when it was drawn from a microbe rather than from human or animal cells, or when it was formed artificially in the test tube as a monolithic double string of a single kind of segment, like poly-A-T or poly-G-C.

A natural thyroid hormone and an artificial female hormone had no effect whatsoever on DNA or DNA-protein.

The scientists feel that the hormones, by allowing strands in certain sections of the DNA to spread apart, behave like a switch in activating the genes. When the genes are turned on, they initiate chemical reactions which make the cells grow, differentiate or specialize, move, produce useful substances, or perform other services. Some of these things the genes may do directly; other things they do by producing more DNA or some RNA, which in turn manufactures enzymes and other highly essential proteins.

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Each WinGel tablet or teaspoon (5 ml.) contains 410 mg. of combined, highly reactive, short polymer, aluminum and magnesium hydroxides **stabilized with hexitol.**

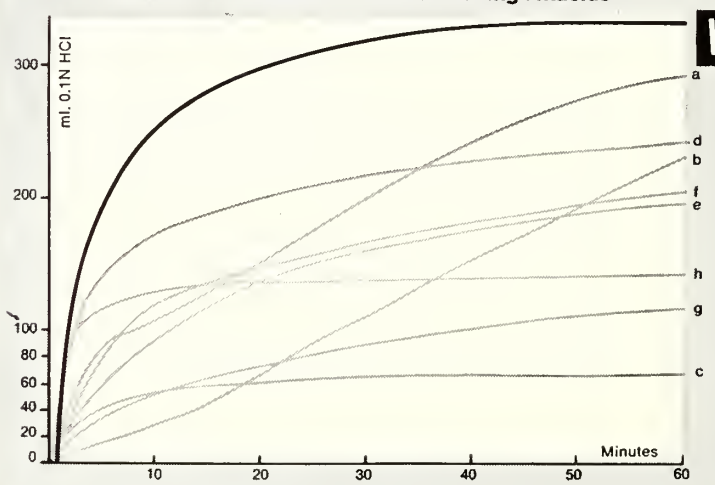
Neutralizes 300 times its active-ingredient weight in gastric acid for fast, long-lasting relief

Gastric or duodenal ulcer, acute or chronic gastritis, gastric hyperacidity...wherever there is "acid overflow" new WinGel can provide faster, longer, more complete neutralization.

In recent laboratory comparisons* with eight other leading antacids, new WinGel tablets not only neutralized more hydrochloric acid per active-ingredient weight, but neutralized it faster and longer than all other antacids tested.

Pleasant pink in color, WinGel is delicately mint flavored with a smooth-as-cream texture—qualities sure to please the patient on long-term therapy. New WinGel is also specially formulated to avoid constipation or diarrhea.

New WinGel neutralizes 300 times its active-ingredient weight in 0.1N hydrochloric acid—neutralizes more acid faster than other leading antacids



Rate of 0.1N hydrochloric acid neutralization at pH 3.5 and 37° with WinGel and eight other leading antacid tablets—*in vitro*. Samples equaled the weight of tablet material containing 1.0 Gm. active ingredients.*

*Hinkel, E. T., Jr. (New York): Data in the files of the Department of Medical Research, Winthrop Laboratories.

Dosage: Peptic ulcer or gastritis—from 2 to 4 teaspoons of WinGel liquid or 2 to 4 tablets chewed or allowed to dissolve in the mouth every two to four hours. **Gastric hyperacidity**—2 tablets or teaspoons about ½ to one hour after meals as needed; children from 7 to 14 years of age, 1 or 2 tablets or 1 or 2 teaspoons of liquid as needed.

How Supplied: Liquid in bottles of 8 fl. oz. and 1 pint. **Tablets** in cellophane strips, boxes of 50 and 100. (One teaspoon of WinGel liquid is equivalent to one WinGel tablet in acid-combining capacity.) WinGel, trademark reg. U.S. Pat. Off.

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Maine Heart Association Notes
116 State Street, Augusta, Maine

Fatal Ventricular Fibrillation During Carotid Sinus Stimulation

. . . Lown and Levine stated that complications of this stimulation such as "permanent cessation of the heartbeat or serious ventricular arrhythmias are almost unheard of." Carotid sinus stimulation is widely and effectively used in the diagnosis and treatment of cardiac arrhythmias.

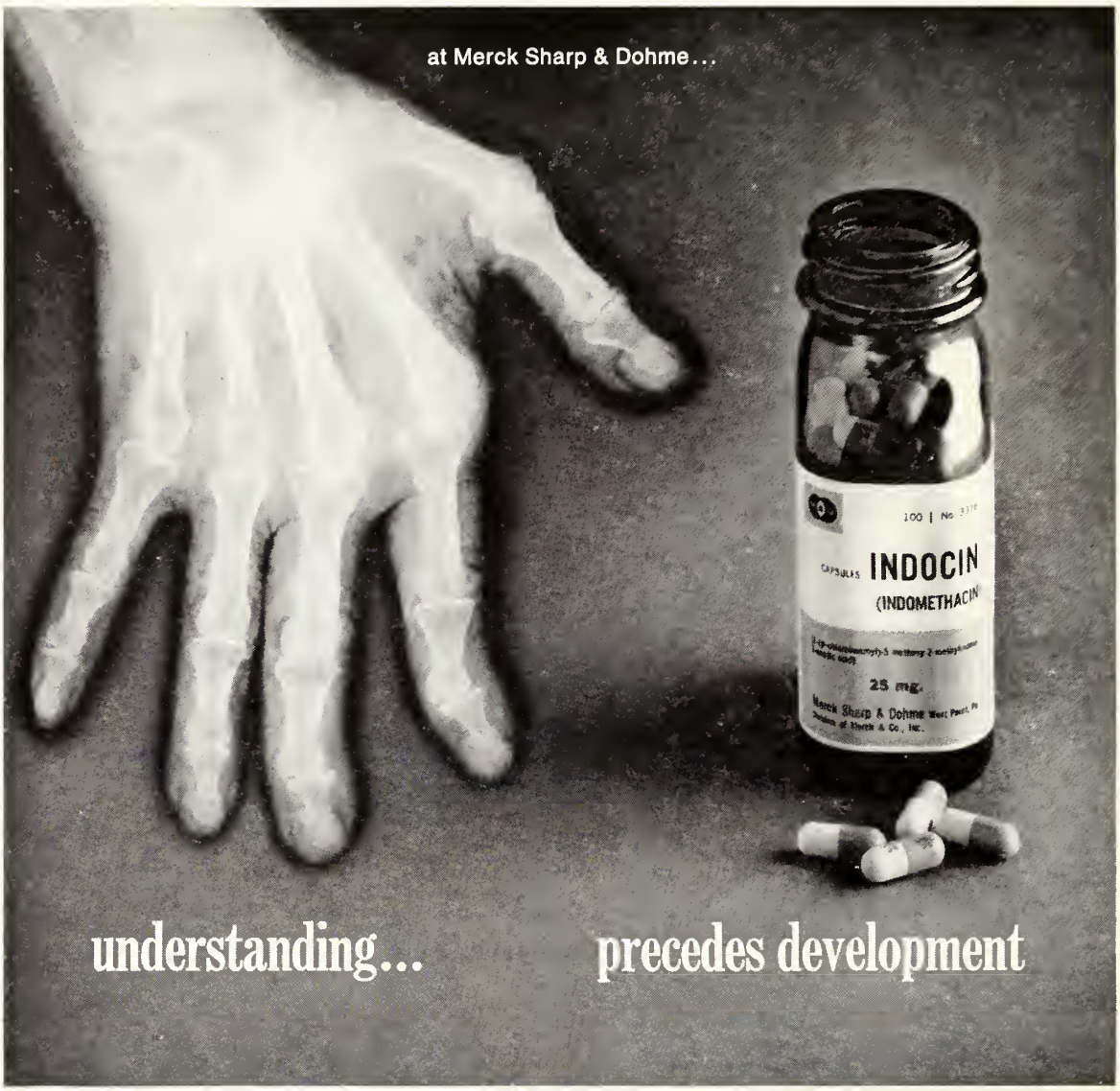
. . . Two later reports have clearly documented ventricular fibrillation occurring as a result of carotid sinus stimulation. . . . The following report describes the third case of ventricular fibrillation occurring during carotid sinus stimulation.

Each of the 3 reported patients in whom ventricular fibrillation developed . . . had been critically ill and had received cardiac glycosides.

The combination of digitalization, tachycardia with possible myocardial anoxia, plus probable increased levels of circulating catecholamines may combine to increase markedly ventricular automaticity. Carotid sinus stimulation, by increasing vagal tone, may slow supraventricular pacemakers or increase the A-V block, thus freeing the ventricle from higher pacemaker control.

Although the great clinical usefulness and the rarity of severe complications occurring with carotid sinus stimulation must be emphasized, caution in its application should still be exercised. Thus, in the critically ill patient receiving digitalis, and particularly in those patients in whom digitalis toxicity is strongly suspected, it would seem wise to employ carotid sinus stimulation with great care. . . . Electrocardiographic monitoring in these cases should be done routinely, and facilities should be readily available for possible electrical, pharmacologic, and perhaps even surgical treatment.

Submitted by Jacob B. Dana, M.D.
Reference: Sidney Alexander, M.D. and Wong Chiu Ping, M.D., The American Journal of Cardiology, Vol. 18, pages 289-291, 1966.



The synthesis of cortisone was accomplished by Merck Sharp & Dohme in 1948—the famous "Compound E" used by Dr. Philip Hench in his historic experiment at the Mayo Clinic.

But proud as we are of our role in the development of cortisone and subsequent corticosteroids, we have continued to seek a greater understanding of arthritic disorders and new drugs for their treatment.

One such drug—INDOCIN® (indomethacin), a nonsteroid, anti-inflammatory agent fundamentally different in structure and activity from other drugs in use—was recently made available for the treatment of arthritic conditions. It opens new possibilities for the long-term management of arthritis and inflammatory disease.

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Indications: Chronic and acute rheumatoid arthritis, rheumatoid (ankylosing) spondylitis, degenerative joint disease (osteoarthritis) of the hip, and gout.
Contraindications: Active peptic ulcer, gastritis, regional enteritis, or ulcerative colitis. Safety in pregnancy has not been established. Not recommended for pediatric age groups.
Warning: Patients who experience dizziness, lightheadedness, or feelings of detachment on INDOCIN should be cautioned against operating motor vehicles, machinery, climbing ladders, etc. Use cautiously in patients with psychiatric disturbances, epilepsy, or parkinsonism.
Precautions and Adverse Reactions: Most commonly, headache, dizziness, lightheadedness, G.I. disturbances. The C.N.S. effects are often transient and frequently disappear with continued treatment or reduced dosage. The severity of these effects may occasionally require cessation of therapy. G.I. effects may be minimized by giving the drug with food or with antacids or immediately after meals. Ulceration of the stomach, duodenum, or small intestine has been reported and, in a few instances, severe bleeding with perforation and death. Gastrointestinal bleeding with no obvious ulcer formation has also been noted; INDOCIN should be discontinued if G.I. bleeding occurs. As a result of G.I. bleeding, some patients may manifest anemia, and for this reason periodic hemoglobin determinations are recommended. Rare reports of effects not definitely known to be attributable to INDOCIN include bleeding from the sigmoid colon (either from a diverticulum or without a known previous pathologic condition), perforation of preexisting sigmoid lesions (diverticulum, carcinoma), and hematuria. In other rare cases, a diagnosis of gastritis has been made while the drug was being given. One patient developed ulcerative colitis, and another, regional ileitis, while receiving INDOCIN; when the drug was given to patients with preexisting ulcerative colitis, there was an increase in abdominal pain. Infrequently observed side effects may include drowsiness, tinnitus, mental confusion, depression and other psychic disturbances, blurred vision, stomatitis, pruritus, edema, and hypersensitivity reactions. Slight BUN elevation, usually transient, has been seen in some patients, although the preponderance of evidence indicates that INDOCIN does not adversely affect renal function, even in patients with preexisting renal disease. Nevertheless, renal function should be checked periodically in patients on long-term therapy. Leukopenia has been seen in a few patients. Transient elevations in alkaline phosphatase, cephalin-cholesterol flocculation, and thymol turbidity tests have been observed in some patients and, rarely, elevations of SGOT values; the relationship of these changes to the drug, if any, has not been established. As with any new drug, patients should be followed carefully to detect unusual manifestations of drug sensitivity.
Before prescribing or administering, read product circular with package or available on request.

News and Notes

Central Maine General Hospital
Fall 1966 Refresher Course
in Arthritis and Rheumatism

Hiebert Hall — 3:00-6:00 P.M.

Oct. 19—*Rheumatoid Arthritis — Pathophysiology, Diagnosis, Clinical Course and Management*
James D. C. Gowans, M.D., Medical Staff Physician and Chief of Arthritis Clinic, New England Center Hospitals, Boston; Assistant Professor of Medicine, Tufts University Medical School

Oct. 26—*Concept of Autoimmune Disease*
Robert S. Schwartz, M.D., Chief of Clinical Immunology Service, New England Center Hospitals; Associate Professor of Medicine, Tufts University Medical School

Collagen Diseases
Robert P. McCombs, M.D., Senior Physician and Chief of Allergy and Pulmonary Service, New England Medical Center Hospitals; Professor of Graduate Medicine, Tufts University Medical School

Nov. 2—*Reiter's Syndrome, Rheumatoid Spondylitis, Psoriatic Arthropathy, Degenerative Joint Disease and Miscellaneous Arthropathies*
John A. Bolzan, M.D., Physician-in-Charge, Arthritis Clinic, St. Elizabeth's Hospital; Assistant Professor of Clinical Medicine, Tufts University Medical School

Nov. 9—*Gout*
Infectious Arthritis
James D. C. Gowans, M.D.

Nov. 16—*Panel on Medical and Surgical Treatment of the Rheumatic Diseases*
James D. C. Gowans, M.D.
George J. Robertson, M.D., Medical Staff Physician, New England Center Hospitals; Medical Director, Bingham Associates Fund; Assistant Professor of Medicine, Tufts University Medical School
Arthur A. Thibodeau, M.D., Chief of Orthopedic Services, New England Center Hospitals; Associate Professor of Orthopedics, Tufts University Medical School

Fee: $25.00. No charge to interns and residents.

State of Maine Board of Registration of Medicine
Secretary — George E. Sullivan, M.D.
Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine
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Roman D. Castellanos, M.D., 77 French St., New Brunswick, N.J.
Jay W. Cha, M.D., Bronx Municipal Hosp., Bronx, N.Y.
Sara K. Cope, M.D., 115 Wayside Rd., Portland, Me.
Raul Correa, M.D., 79 Wilson Ave., Framingham, Mass.
Juan F. Gaeta, M.D., Roswell Park Mem. Inst., Buffalo, N.Y.
Parviz Janfaza, M.D., 580 Willard St., Quincy, Mass.

Mamoru Kaneko, M.D., 372 Central Park West, New York, N.Y.
Hae A. Kim, M.D., 85 E. 43rd St., Brooklyn, N.Y.
Young K. S. Kim, M.D., 753 Mendon Rd., Woonsocket, R.I.
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Soleiman Mohamadian, M.D., 3579 Rt. 46, Parsippany, N.J.
Anthony C. Nassif, M.D., 651 Elm St., Buffalo, N.Y.
Alexander T. Parkinson, M.D., Pittsfield Mass. Gen. Hosp., Pittsfield, Mass.
Giselle S. Pechet, M.D., 76 Kenilworth St., Newton, Mass.
Dimitri G. Polydefkis, M.D., Augusta State Hosp., Augusta, Me.
Yvan J. Silva, M.D., Royal Victoria Hosp., Montreal, Can.
Joseph H. S. Suh, M.D., 438 Parker St., New Bedford, Mass.
Cyril Toker, M.D., Mount Sinai Hosp., New York, N.Y.

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Simon Meleck, M.D., 385 Lincoln St., New Britain, Conn.
Walter A. Mendyka, M.D., Machias, Me.
Manuel Posso, M.D., 133 Kenmore Dr., Danville, Va.
Robert L. Shelton, M.D., 135 Diamond St., New Haven, Conn.

Book Review

Blood Diseases of Infancy and Childhood by Carl H. Smith, M.A., M.D. Published by the C. V. Mosby Company, St. Louis, 1966. 800 pages with 86 illustrations. Price $22.00.

Doctor Smith's new (2nd) edition of this book is a remarkable compendium of information touching on subjects related to blood diseases as well as covering all of the facets of the ills that blood and its component parts are heir to.

His opening chapters are concerned with blood development and he discusses in detail the maternal-fetal interaction and their immunological relationships. He then breaks the contents into the various cells and their formations: Normal and Pathological. As an example: under ERYTHROCYTES details are given of the heme derivation as well as the usual and unusual erythrocyte, its diseases and its relation to beta lipoproteins.

From this point he then enlarges on blood terms, groups, genetics and his points are ideal for blood bank technique and further specifics are given in agglutination as: O group by anti-H sera, and so forth, which is of both clinical and laboratory significance.

After some general discussion of anemias he spends considerable time, in fact complete chapters, on specific anemia types, example: iron deficiency, megaloblastic and hemolytic. Entities of the many hemoglobinopathies and methods of separation by electrophoresis are considered in detail. Even the use in children of $Chromium_{51}$ and $Iron_{59}$ in red cell survival and the DFP_{32} in white blood cell labeling is suggested and comparisons are made with adult survival times. Thus in many directions this book in its diagnostic approaches is as applicable to adults as to children. But on the other hand, the needs and necessities of transfusions are at variance with adult criteria and the problems of limitations and hazards of overloaded circulation, reactions to protein material as well as the prevention and treatment with drugs is expressed more in terms of the child.

In chapters on jaundice, erythroblastosis and other causes are displayed, again with treatment. I am a little surprised at his use here of the "Direct and Indirect Bilirubin," as most workers now mention the inaccuracy of this method and prefer the break-down into free bilirubin and the mono and diglucuronides. However, it is well to mention that excellent references are catalogued with each chapter and many quotations are present from work completed within the past year.

Again with the leucocytes as with the red cells, Doctor Smith outlines descriptions of cells, enzyme systems, oxygen requirements and the necessities for growth and net result of cell destruction. Functional activities and antibody formation round this out to relate the cell to the body as a whole and to diseases of the whole blood system.

Perhaps the most disappointing aspect of the book is encountered in the white cell group of diseases. Here, the absence of colored plates, true throughout the book, is felt most strongly; for thousands of word pictures of cells cannot replace the color pictures. A color plate atlas used in conjunction with this book is a necessity for any laboratory and should extend to the marrow, L. E. preparations, and so forth, as well as to the peripheral blood.

In further chapters on the leucocytes, diseases are enumerated and described in relation to various white blood cells as: eosinophile, plasma cell, Türk's cell and so forth. Even the hereditary variance in cells and effects of viral or immunological reactions are listed. It is of interest that of the modern screening tests, as formalized horse red cells in Infectious Mononucleosis or the ox cell hemolysis, Doctor Smith makes no comment concerning the efficacy of these particular tests. Some emphasis and comment is made, however, on the genetics of both red cell and white cell diseases and methods of tracing them.

Therapy is included in many chapters, but of very recent import is the listing of Folic Acid Antagonists, Aminopterin, Methatrexate and other anti-leukemic agents. Table 21 is an excellent summary. This, along with the chapter on LEUKEMIA, ALLIED DISORDERS, covers the more bizarre entities as: myeloproliferative syndrome, agnogenic myeloid metaplasia, and the group of lymphosarcomas and allied diseases.

The book strays a bit afield from pure blood disorders in the studies of the reticulo-endothelial system, lipid disorders, splenic enlargement and so forth. Under therapy of the latter, he describes the blood vessel surgery, its success areas and its complications as well as other therapeutic measures. The use of chemo-therapeutic agents in the Letterer-Siewe group is also considered.

The late chapters on BLOOD COAGULATION AND PURPURAS may have most appeal to technicians and pathologists in the small hospital though specific areas of therapy for purpuras are given to help clinicians in following such cases. However, more time and words are given to positive factors, heredity and allergy which are of significance to the laboratory as well as the pathological details which interest the research student but may not appeal greatly to the active practitioner.

In all, this is a very "up-to-date" significant book, of value to both clinicians and laboratory workers, and my only real complaint is the lack of colored plates.

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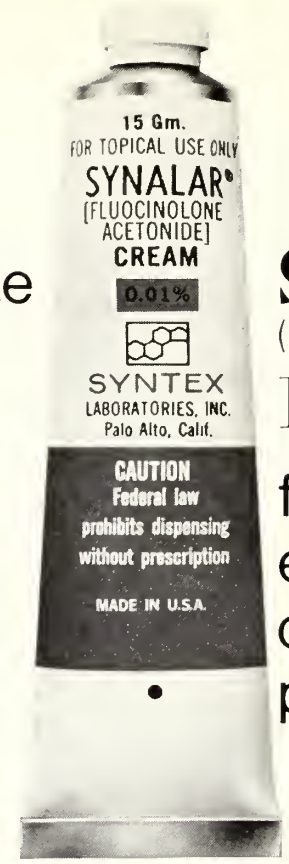
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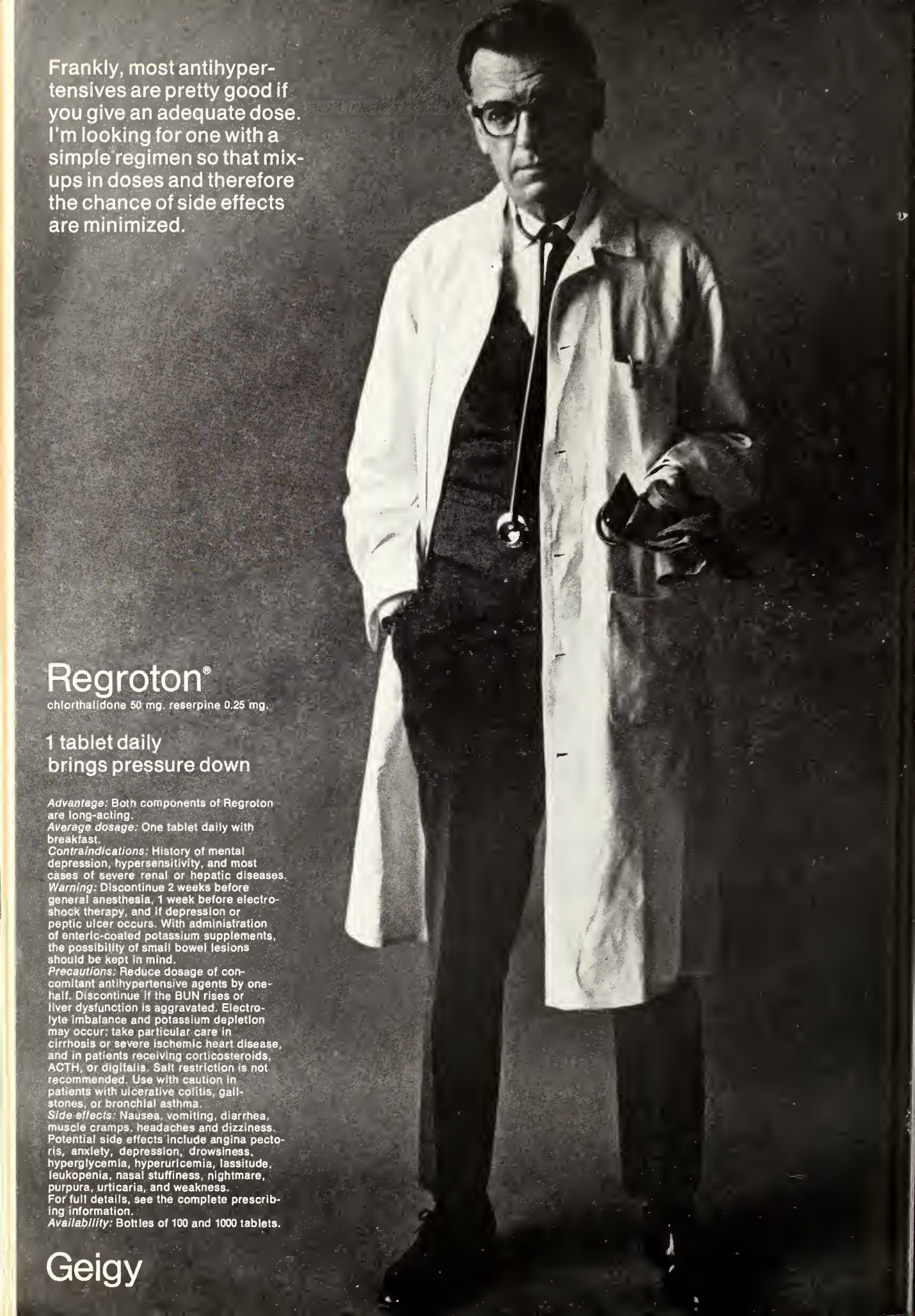


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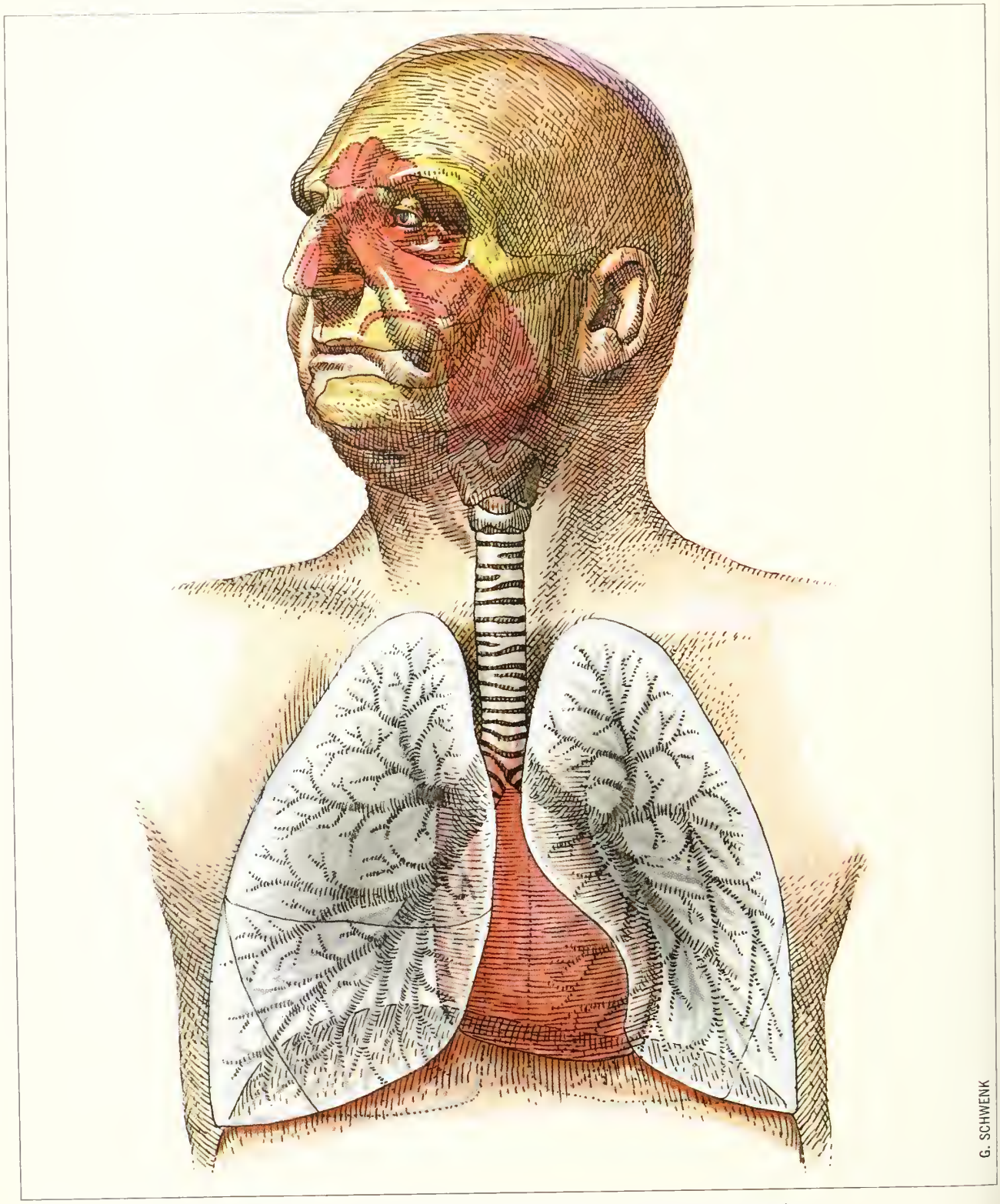
A journal within a journal published quarterly in the interests of better medicine by Dorsey Laboratories, a division of The Wander Company, Lincoln, Nebraska. Address communications to Raymond C. Pogge, M.D., Director of Medicine.



this issue: the common cold and the aging patient

the common cold and the aging patient

Louis J. Vorhaus, II, M.D., F.A.C.P.



Effects of aging on the anatomic and physiologic aspects of the respiratory apparatus.

The chest becomes more fixed, less mobile and less elastic as the bronchial walls and thoracic ligaments lose elasticity. The diaphragm and intercostal muscles atrophy and weaken. The lungs become smaller, flabbier and weigh less, decreasing vital and total lung capacity, increasing residual volume and the alveolar dead space.

Sir William Osler described pneumonia as the welcome friend of the aged patient, because the patient with pneumonia usually died quietly. But today, the well-informed physician is an even better friend of the aging patient, since it is better to live than to die, no matter how quietly.

One of the first avenues of approach in the control of the hazards of respiratory disease in the aging patient is prompt and proper attention to the common cold or upper respiratory infection. The common cold may be the first step in the relatively short path to lower respiratory infection, broncho-pneumonia and death. This train of events occurs frequently among older persons. Indeed, pneumonia is one of the most common causes of their admission to hospitals and ranks high on the list of geriatric killers. Colds are more debilitating in elderly people and the aged are more likely candidates for secondary infections such as sinusitis and bronchitis. These infections, in turn, are more prone to lead to bronchopneumonia, because of lowered resistance and anatomic and physiologic changes in the lungs of the elderly.

What is different about the respiratory tree of an aged person and that of an otherwise healthy younger adult? Aging certainly takes its toll on all parts of the body, affecting both anatomic and physiologic aspects of the respiratory apparatus. These changes are in part due to the wear and tear that occurs over the years; the repeated bouts of respiratory infection, long exposure to atmospheric pollutants, to occupational inhalants, smoking, malnutrition, obesity, inactivity and the development of other diseases which may affect the lungs.

With the passage of years, the lungs change. They become scarred and emphysematous and lose their compliance. The whole chest becomes more fixed, less mobile and less elastic.

The anatomic changes that occur in aging render the lungs less efficient. Tests of pulmonary function in senescence show a deterioration characterized by a decrease in vital capacity and total lung capacity, an increase in residual volume and alveolar dead space. Maximum breathing capacity is reduced and uniformity of ventilation deteriorates. These problems are often aggravated by the obstructed breathing, fever and secondary infection associated with the common cold, placing an additional stress on the entire cardiopulmonary reserve.

In addition, the efficiency of a cough is below par in older persons even though they are in good health. This is partly due to the decreased respiratory excursions and distensibility of the chest wall, and partly from loss of elasticity of bronchial walls which tend to make them collapse in a cough.

The elderly patient's resistance to infection is often reduced. Nutritional deficiencies are more common in aged people. There is some evidence to indicate that their capacity to respond to stress is less efficient. Finally, there is often relatively meager symptomatic response to acute disease. The absence of obvious or dramatic clinical signs and symptoms of severe illness is particularly dangerous because, coming as it

(concluded on following page)

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does in a person whose defenses are weakened both locally and systemically, pulmonary disease may progress rapidly to irreversible stages before medical attention is sought. Respiratory infection is especially hazardous because the aged patient responds badly to hypoxia. Not only is his response to oxygen lack impaired, but the work of breathing, due to decreased compliance of the lung and increased stiffness of the thorax, is markedly augmented.
Many patients late in life are in a precarious and delicate cardiopulmonary balance which is easily decompensated from relatively minor insults such as colds and upper respiratory infections.

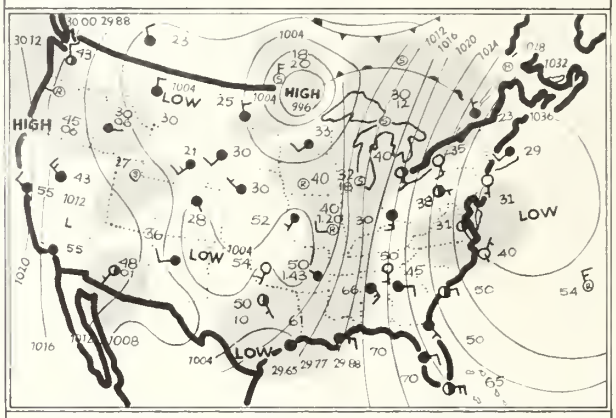
For all of these reasons, geriatricians long have stressed the importance of preventing respiratory insults. Today we have better ways of treating respiratory infection, improved techniques for clearing the lungs and bronchial tubes of secretions and better understanding of ways of improving ventilation. We possess a broader spectrum of antimicrobial agents including newer ones to deal with previously resistant organisms. Even so, the death rate from pneumonia is high in older people, and it is preferable to avoid the disease than to treat it. To do so, attention must be paid to the general maintenance of good health and all that implies, as well as to the prevention, elimination and treatment of associated conditions that predispose to or cause pneumonia such as chronic upper or lower respiratory infection, respiratory allergy, chronic sinusitis and exposure to inspired irritants.

Colds and other minor respiratory infections, which favor the development of broncho-pneumonia, should be treated vigorously and promptly, particularly those patients whose aging process has been accompanied by the development of chronic pulmonary disease. Upper respiratory passages should be cleared with decongestants. Sinuses should be drained adequately. And, when indicated, appropriate antimicrobial therapy should be instituted before serious infection of the lower respiratory tree supervenes.

In decades past it was understandable that physicians welcomed pneumonia for the aged patients because it offered them a quiet and peaceful demise. Today we recognize that in many cases, peaceful as it may have been, such deaths were often avoidable. With the current knowledge and understanding of the problems that respiratory infections impose on aging people, vigilant medical attention can often restore them to a vigorous, rewarding and productive life so that the many opportunities that exist today for people to enjoy their golden years are realized and not stolen by untimely death.

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You might also say that all interns aren't alike, either.





SQUIBB NOTES ON THERAPY



Breast-feeding and the "modern mother"

Despite a mild resurgence of interest in the importance of breast-feeding a few years ago, many women today do not choose to nurse their young. This is for a variety of reasons—social, economic, cultural and sometimes medical. In such cases the physician's task is to find the most suitable means of preventing lactation and easing the pain of breast engorgement.

The means of therapy

The value of hormone therapy for this indication is of course well established. Both androgen and estrogen are known to inhibit the production and secretion of the lactogenic hormone by the anterior pituitary. As estrogen levels decline sharply at parturition, lactogenesis is established. When androgen and estrogen are administered to the patient before the release of the lactogenic hormone lactation and breast engorgement are usually prevented.

The time of therapy

The time of administration of this combined medication is crucial; it must be given early enough to suppress the pituitary prolactin and last long enough to permit physiologic readjustment during the puerperium. Excellent results are most often seen when therapy is administered before the onset of the second stage of labor.

However, factors other than effectiveness must also be considered. The agent selected should not interfere in any way with parturition, subsequent uterine involution and the restoration of normal ovarian cyclic function. Furthermore, it should not cause rebound breast engorgement or other manifestations of hormonal imbalance.

A balanced formulation

Providing single-dose therapy for the prevention of lactation and breast engorgement, Deladumone OB is a potent androgen-estrogen combination with a prolonged action. The optimal balance of androgenic and estrogenic hormones achieved in this preparation minimizes the disadvantages inherent in single hormone therapy, such as rebound breast engorgement. Involution of the uterus and resumption of menstrual cycles are not affected.

As reported in a recent published study (Roser, D. M.: Obstet. & Gynec. *27*:73, 1966), Deladumone OB provided good suppression of breast engorgement in 95.3% and suppression of lactation in 81.1% of 86 obstetrical patients. These results are in general agreement with those of many earlier investigations; in several studies this injectable androgen-estrogen combination proved to be superior to oral medication.

Dosage:

As a single injection of 2 cc. before the onset of the second stage of labor.

Contraindications:

Established or suspected mammary cancer or genital malignancy.

Precautions and Side Effects:

Certain patients may be unusually responsive to either estrogenic or androgenic therapy. In such individuals virilization, uterine bleeding or mastodynia may occur.

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Deladumone OB, providing 180 mg. testosterone enanthate and 8 mg. estradiol valerate per cc., is available in 2 cc. Unimatic® disposable syringes and in 2 cc. vials. Both preparations are dissolved in sesame oil, with 2% benzyl alcohol as a preservative. *Before use, consult product literature for full prescribing information.*

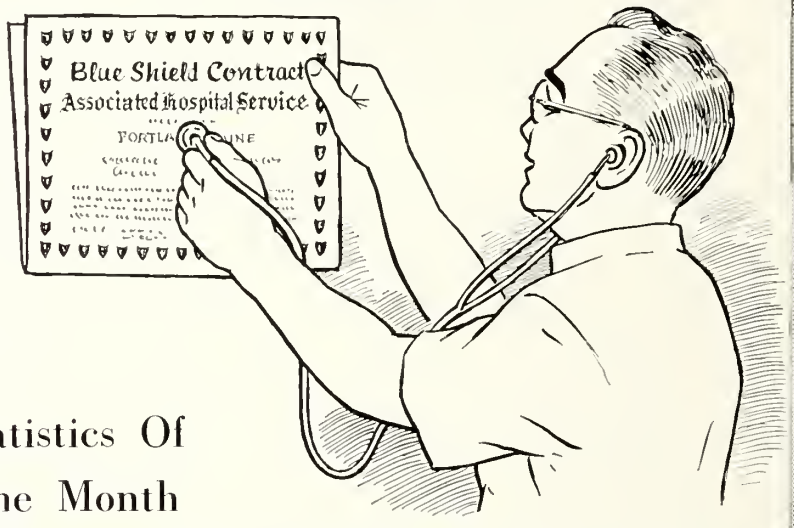
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Of the medical care claim dollars, 86 percent went for in-hospital cases and 14 percent for home and office cases. The average in-hospital medical care claim was $43.44; the office and/or home claim averaged $6.41.

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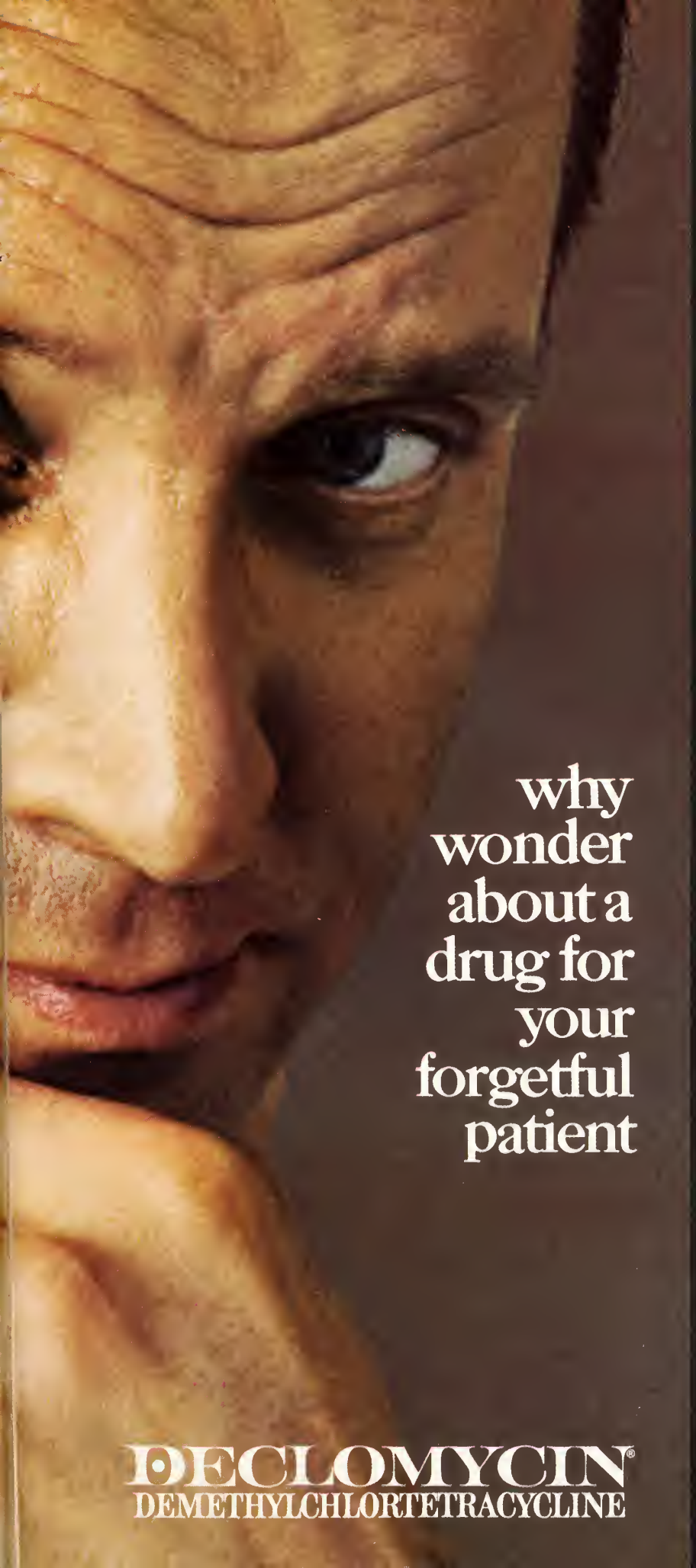
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Precautions and Side Effects—Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

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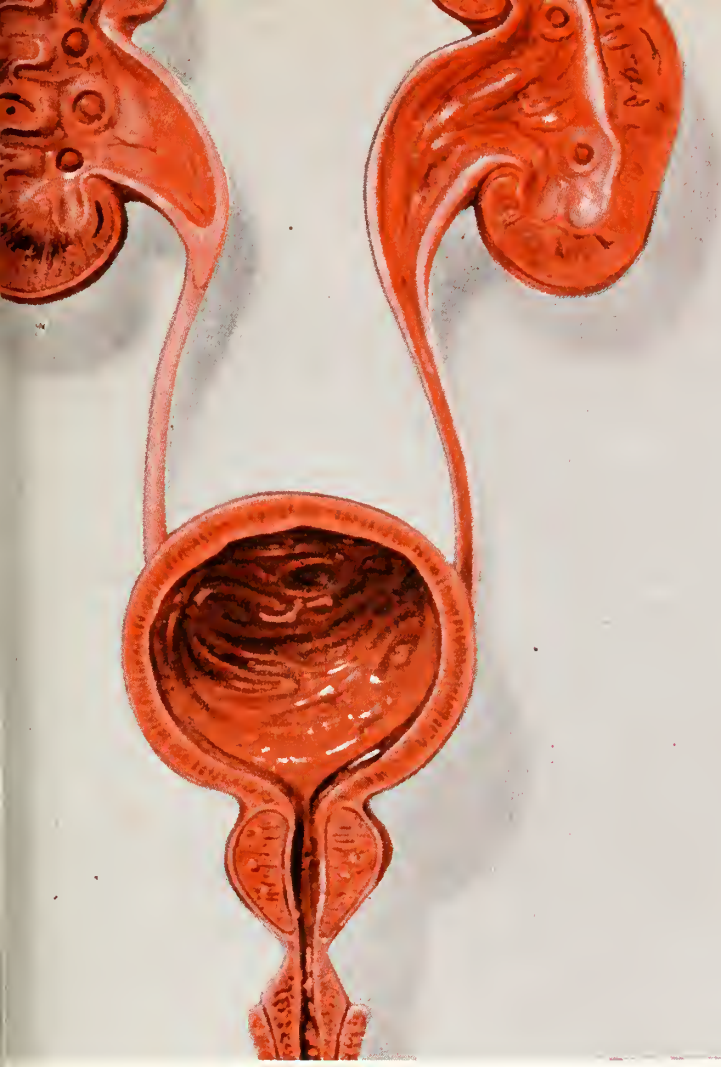
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pyelonephritis?
pyelitis?
urethritis?
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in any case,
usually gram-negative*

Therapy:

two 500 mg. Caplets® q.i.d.
(initial adult dose)

Indications: Urinary tract infections caused by gram-negative and some gram-positive organisms.

Side effects: Mainly mild, transient gastrointestinal disturbances; in occasional instances, drowsiness, fatigue, pruritus, rash, urticaria, mild eosinophilia, reversible subjective visual disturbances (overbrightness of lights, change in visual color perception, difficulty in focusing, decrease in visual acuity and double vision), and reversible photosensitivity reactions. Marked overdosage, coupled with certain predisposing factors, has produced brief convulsions in a few patients.

Precautions: As with all new drugs, blood and liver function tests are advisable during prolonged treatment. **Pending further experience, like most chemotherapeutic agents, this drug should not be given in the first trimester of pregnancy. It must be used cautiously in patients with liver disease or severe impairment of kidney function.** Because photosensitivity reactions have occurred in a small number of cases, patients should be cautioned to avoid unnecessary exposure to direct sunlight while receiving NegGram, and if a reaction occurs, therapy should be discontinued. The dosage recommended for adults and children should not arbitrarily be doubled unless under the careful supervision of a physician. Bacterial resistance may develop.

When testing the urine for glucose in patients receiving NegGram, Clinistix® Reagent Strips or Tes-Tape® should be used since other reagents give a false-positive reaction.

Dosage: Adults: Four Gm. daily by mouth (2 Caplets® of 500 mg. four times daily) for one to two weeks. Thereafter, if prolonged treatment is indicated, the dosage may be reduced to two Gm. daily. Children may be given approximately *25 mg. per pound* of body weight per day, administered in divided doses. The dosage recommended above for adults and children should not arbitrarily be doubled unless under the careful supervision of a physician. Until further experience is gained, infants under 1 month should not be treated with the drug.

How supplied: Buff-colored, scored Caplets® of **500 mg. for adults,** conveniently available in bottles of 56 (sufficient for one full week of therapy) and in bottles of 1000. **250 mg. for children,** available in bottles of 56 and 1000.

References: (1) Based on 23 clinical papers, 1512 cases. Bibliography on request. (2) Bush, I. M., Orkin, L. A., and Winter, J. W., in Sylvester, J. C.: Antimicrobial Agents and Chemotherapy—1964, Ann Arbor, American Society for Microbiology, 1965, p. 722.

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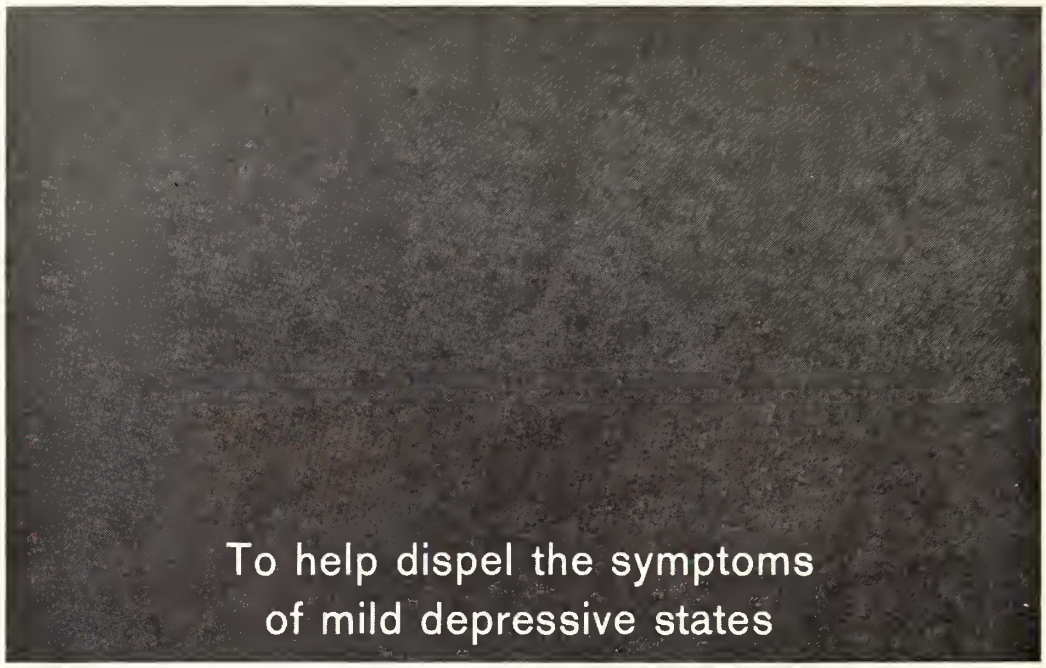
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eradicates most urinary tract infections...

- Low incidence of untoward effects; no fungal overgrowth, crystalluria, ototoxic or nephrotoxic effects have been observed.

- "Excellent" or "good" response reported *in more than 2 out of 3* patients with either chronic or acute gram-negative infections.[1]

*As many as 9 out of 10 urinary tract infections are now caused by gram-negative organisms: E. coli, Klebsiella, Aerobacter, Proteus, Paracolon or Pseudomonas[2]... However, infections of the urethra and prostate caused by non-gonococcal gram-negative organisms are believed to be less prevalent.



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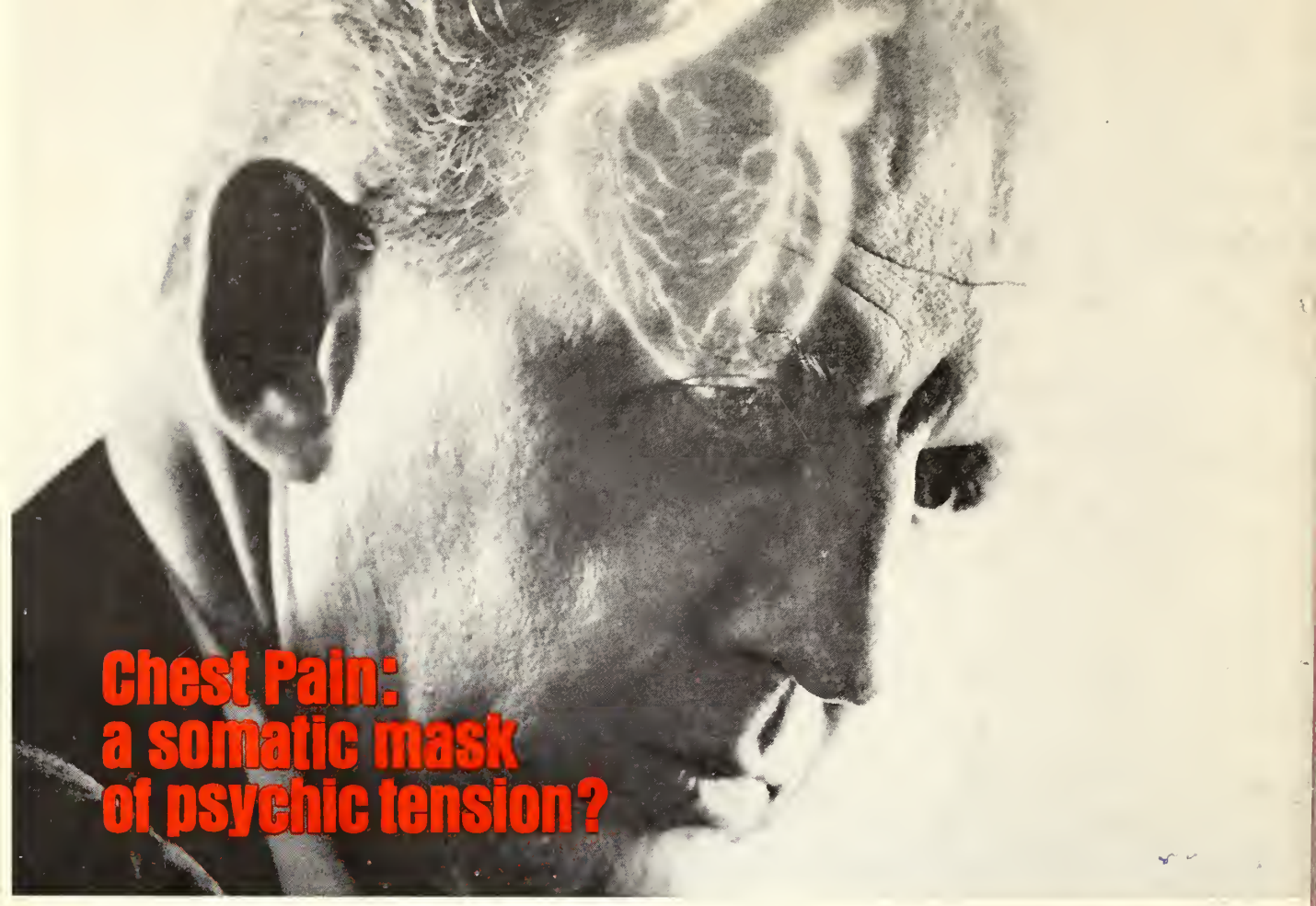
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Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage — Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hrs, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

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VOLUME 57 NOVEMBER, 1966 NUMBER 11

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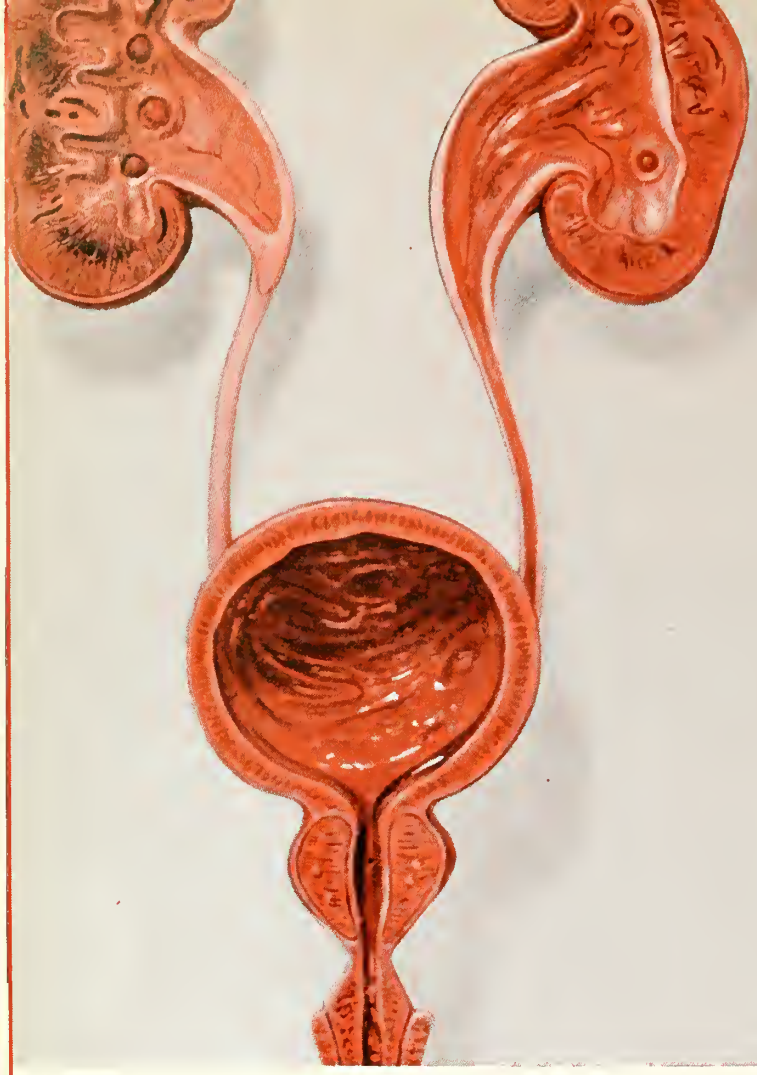
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pyelonephritis?
pyelitis?
urethritis?
prostatitis?

in any case,
usually gram-negative*

Therapy:

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(initial adult dose)

Indications: Urinary tract infections caused by gram-negative and some gram-positive organisms.

Side effects: Mainly mild, transient gastrointestinal disturbances; in occasional instances, drowsiness, fatigue, pruritus, rash, urticaria, mild eosinophilia, reversible subjective visual disturbances (overbrightness of lights, change in visual color perception, difficulty in focusing, decrease in visual acuity and double vision), and reversible photosensitivity reactions. Marked overdosage, coupled with certain predisposing factors, has produced brief convulsions in a few patients.

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- "Excellent" or "good" response reported *in more than 2 out of 3* patients with either chronic or acute gram-negative infections.[1]

*As many as 9 out of 10 urinary tract infections are now caused by gram-negative organisms: E. coli, Klebsiella, Aerobacter, Proteus, Paracolon or Pseudomonas[2]... However, infections of the urethra and prostate caused by non-gonococcal gram-negative organisms are believed to be less prevalent.

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CONTRAINDICATIONS: Pyloric obstruction, gastric retention, obstructive organic disease of the gastrointestinal troct, orgonic cordiosposm, duodenal stenosis, stenosing peptic ulcer, and urinary blodder neck obstruction or prostatic hypertrophy ore controindicotions to the use of this drug.

WARNING: Overdosoge produces anticholinergic side effects. Although the onimal reproduction studies are negative, until there is clinical confirmation of safety in pregnancy, this product should not be used in women who may become pregnant unless in the opinion of the physicion the benefits outweigh the risks.

PRECAUTIONS: Use only with caution in potients with certain types of cordiovascular disease, since anticholinergic drugs may cause arrhythmios. Extensive clinical studies of TREST have shown no evidence of glaucoma. However, the possibility exists thot it can occur since it has been reported os o characteristic side effect of anticholinergic drugs. This product does not replace definitive treatment in organic gastrointestinol diseose.

SIDE EFFECTS: Side effects ore generolly obsent when TREST is used in the recommended dosage of 1 to 2 mg. three times daily. Uncommonly, allergy or sensitivity to the drug may be manifested by generolized rash or widespread desquamation. In case of prolonged or mossive overdosage, dry mouth, blurred vision, and urinary retention, typical side effects common to anticholinergic drugs, moy occur. Occasionally, sensitive patients may notice mild dryness of mouth or slight blurring of vision from doses of 2 mg. or more. Most patients tolerote single doses of 5 mg. without such side effects.

DOSAGE: The usual adult dosage is 1 mg. by mouth three times daily. If necessary, the dose may be increased to 2 mg. three times daily. Pediatric dosage has not been determined.

CAUTION: Federal law prohibits dispensing without prescription.

Puts the G.I. tract to rest

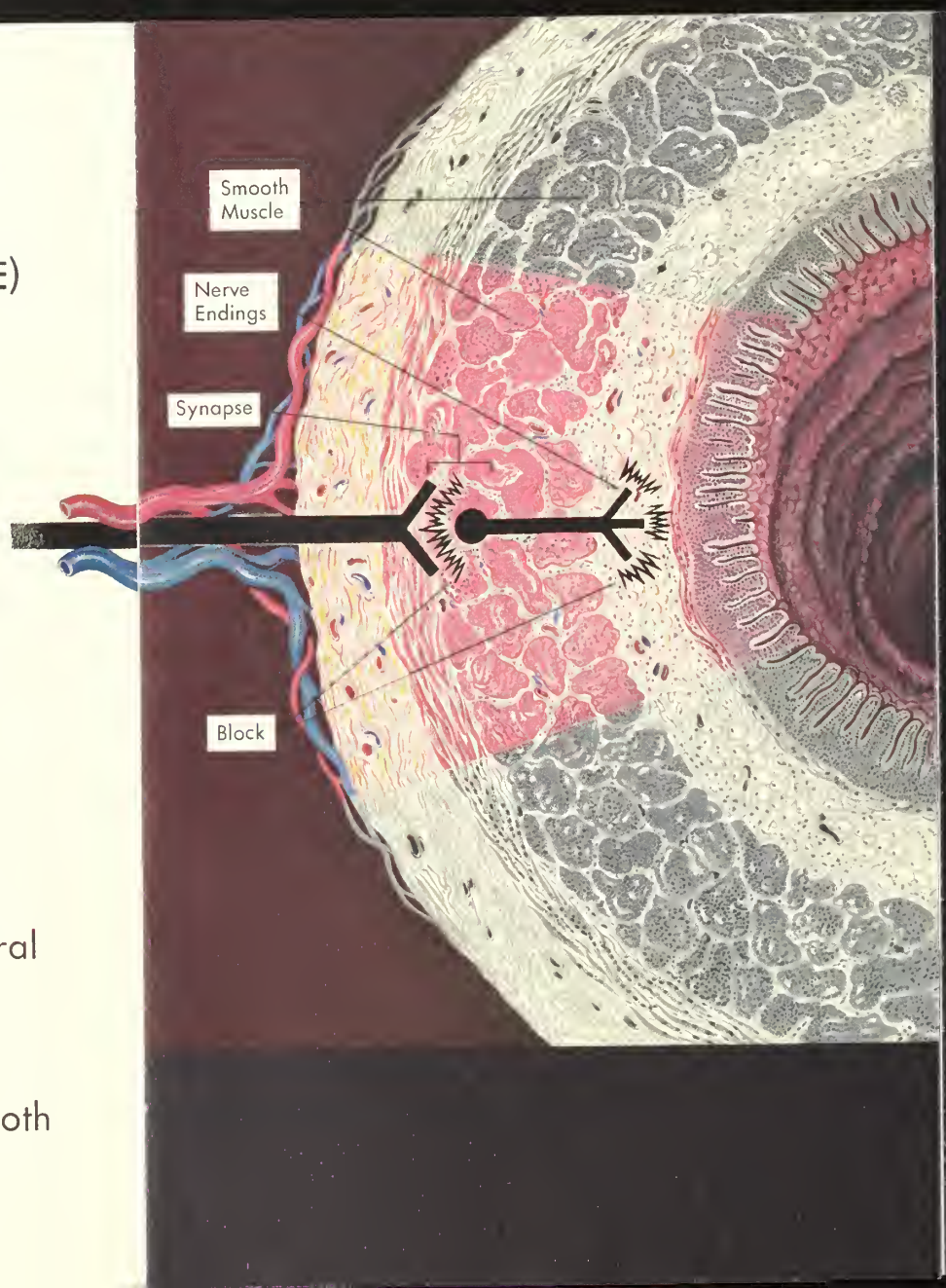
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4848 Printed in U.S.A. 10/66

An antispasmodic that antagonizes.

How TREST®
(METHIXENE HYDROCHLORIDE) works.

parasympathetic nerve

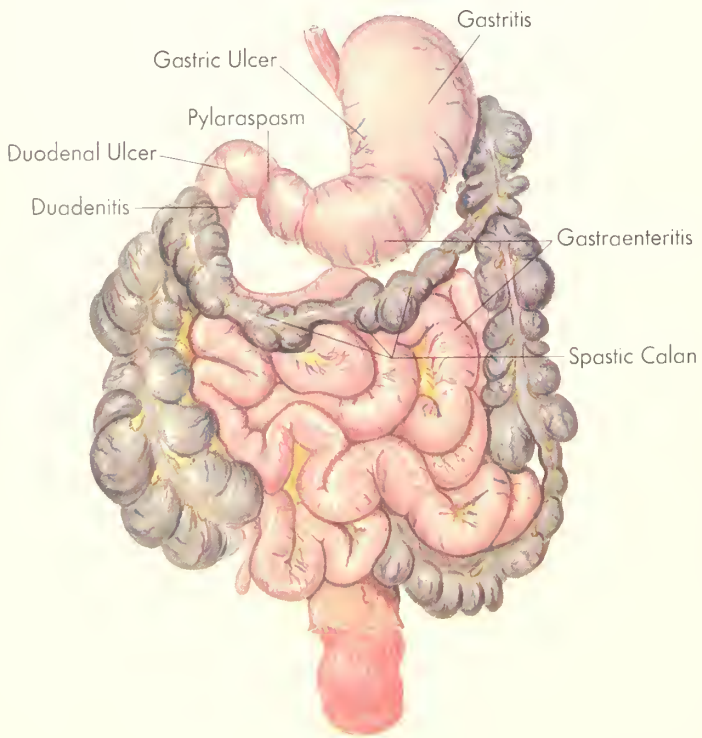


TREST (methixene hydrochloride) directly antagonizes the parasympathetic nervous system.

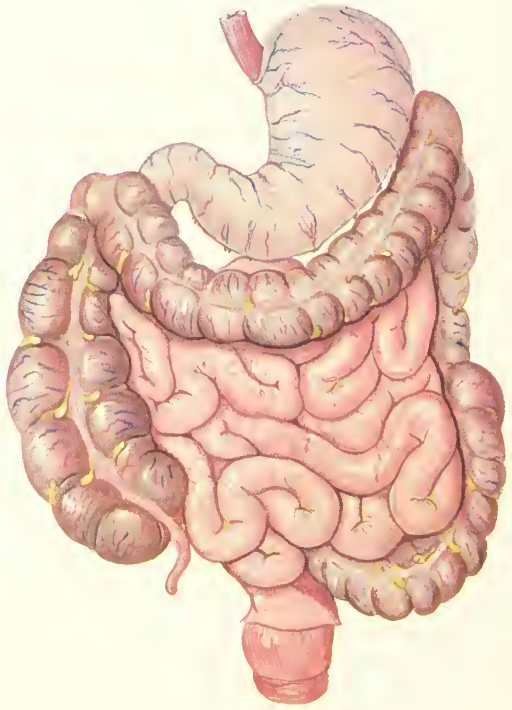
TREST (methixene hydrochloride) blocks the action of acetylcholine formed at the synapses and visceral parasympathetic nerve endings.

Therefore, nerve impulses are prevented from reaching the smooth muscle layer.

TREST® (METHIXENE HYDROCHLORIDE)
relieves gastrointestinal spasm, hyperactivity and associated pain in:



Put the gut to rest
with your prescription for
TREST®
(METHIXENE HYDROCHLORIDE)



Evidence that TREST®
(METHIXENE HYDROCHLORIDE) works.

EFFECTIVENESS AND SAFETY
t.i.d. dosage in milligrams

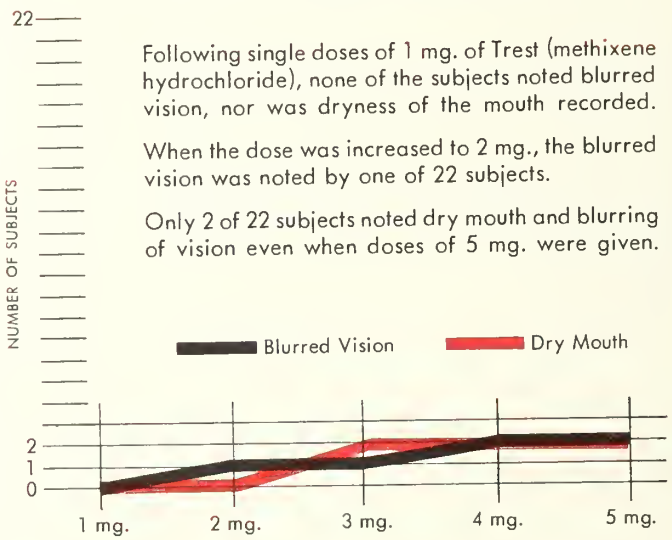
1 mg. symptomatic relief

2 mg. upper recommended clinical dose/greater pharmacologic activity and symptomatic relief/side effects uncommon

10 mg.

Studies with 372 subjects revealed no adverse effect upon vital organs when dosage was increased 10 times the normally effective dose of 1 mg. t.i.d. for periods up to 2½ years of continued administration.

LACK OF SIDE EFFECTS



Following single doses of 1 mg. of Trest (methixene hydrochloride), none of the subjects noted blurred vision, nor was dryness of the mouth recorded.

When the dose was increased to 2 mg., the blurred vision was noted by one of 22 subjects.

Only 2 of 22 subjects noted dry mouth and blurring of vision even when doses of 5 mg. were given.

Conclusion: Typical atropine-like side effects are not expected when Trest is used 1 mg. t.i.d. These effects are uncommon when the dosage is increased to 2 mg. t.i.d.

"A highly satisfactory symptomatic response was obtained in 20 of the 23 patients who took 1 mg. of methixene hydrochloride (Trest) by mouth three times daily. No side effects occurred at this dosage during administration of the medication itself.

"Methixene hydrochloride (Trest) provides highly gratifying symptomatic relief in a variety of conditions associated with gastrointestinal motility without producing the usual atropine-like side effects and without requiring concurrent barbiturate sedation." —Martins, J. K.: Clin. Med. 72:1313-1316 (Aug.) 1965.

". . . in a series of 47 patients suffering from various types of functional bowel distress. The superiority of this drug (Trest) in dosage of 1.0 mg. three times daily by mouth over the placebo is statistically significant. At that dosage level, side effects were not observed." —Hufford, A. R.: Clin. Med. 72:1151-1155 (July) 1965.

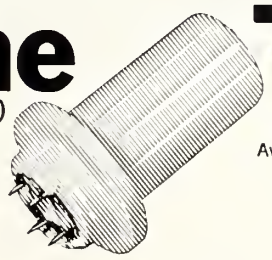
what time is it?

For the past
two years
there's been
one new case
of active tuberculosis
reported for every
four thousand
of U.S. population.

it's time to tine.

Tuberculin, Tine Test

(Rosenthal) Lederle

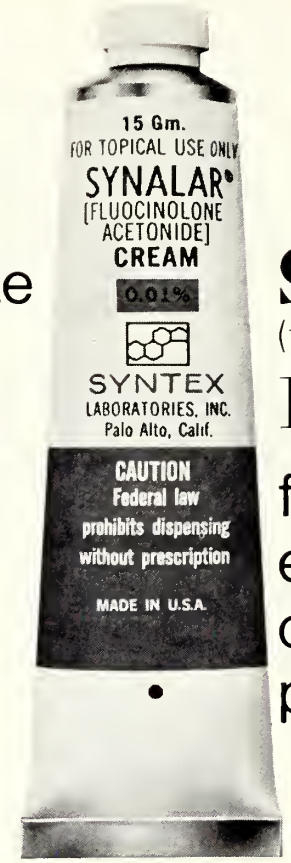


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Synalar® 0.0
(fluocinolone acetonide) **crea**
15 Gm.

for even greater economy in office or hospital practice

the superiority
topica
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Now you can prescribe as little or as much Synalar Cream 0.01% as is needed for a particular therapeutic problem in a size that permits the greatest economy for your patient. The new 15 Gm. tube, for example, is best suited for short-term therapy and for small sites. For more extensive body areas prescribe the 45 Gm. tube—a size that's also ideal for your treatment table. And the 120 Gm. jar is most economical for hospital use. Thus, with Synalar Cream 0.01%, you have the superiority of a modern topical corticosteroid shown to be more effective than 1% hydrocortisone[1-3] plus the economy that makes therapy practical for use in more dermatologic conditions, in long-term maintenance, with occlusive dressings in resistant cases, and in extensive area involvement.

Contraindications: Tuberculous, fungal, and most viral lesions of the skin (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of its components. **Precautions:** 1. *General*—Synalar Cream 0.01% is virtually nonsensitizing and nonirritating. Where severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. While topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use on p[re]females has not absolutely been established. Therefore, they should[n]used extensively on pregnant patients, in large amounts, or for pr[o]periods of time. 2. *Occlusive dressing method*—With occlusion of e[x]areas, systemic absorption of the corticosteroid may occur, and precautions should be taken. Occasional patients may show conta[c]tivity to a particular dressing material or adhesive. Miliaria, follic[u]pyodermas have been seen infrequently with the use of this techniq[ue]development of infection requires appropriate antibacterial therapy continuation of the occlusive dressing method. Local atrophy an[d]have been reported with protracted occlusive dressing therapy. Whil[e]relapses can be expected to occur in many psoriatic patients, rem[is]may persist for several weeks to several months in favorable cas[es]patient whose psoriasis is in an active stage, with recent appearanc[e]lesions, may not be a good candidate and may show early relaps[e]plastic films may be flammable, and due care should be exercised use. Similarly, caution should be employed when such films are us[ed]left near children to avoid the possibility of accidental suffocati[on]**Effects:** Side effects are not ordinarily encountered with topically corticosteroids. As with all drugs, however, a few patients may re[act]vorably to Synalar under certain conditions. **References:** 1. Cahn, M.[M.], Levy, E. J.: J New Drugs 1:262 (Nov.-Dec.) 1961. 2. Meenan, F. O.[C.], Med Ass 52:75 (Mar.) 1963. 3. Robinson, H. M., Jr., Raskin, J., and [Dill]W. J. R.: Southern Med J 56:797 (Jul.) 1963.

of a modern corticosteroid economy of hydrocortisone

Now...a choice of 3 economical sizes



120 Gm. jar 15 Gm. tube 45 Gm. tube

fluocinolone acetonide — an original steroid from

SYNTEX

LABORATORIES INC., PALO ALTO, CALIF.



Tareyton...with the taste worth fighting for

America's largest-selling charcoal-tip cigarette

©The American Tobacco Company



this part for diarrhea

Kaolin exerts a demulcent action along the gastrointestinal tract and a detoxifying action in the intestines to diminish irritation of the mucosa and lessen hyperperistalsis, nausea and diarrhea.

Pectin exerts its demulcent effect in the entire tract and its detoxifying action primarily in the large bowel to diminish irritation of the mucosa and help restore normal intestinal flora and function.

this part for its discomforts

Belladonna alkaloids as in Donnatal® relieve hypermotility of smooth muscle in the gastrointestinal tract to help control cramping, nausea, and painful straining. Many clinicians consider the belladonna components of Donnagel® to be medicine's most effective depressants of intestinal motility.[1,2] A preparation containing only kaolin and pectin, on the other hand, has "little or no effect on cramps simply because it does not include an agent with antispasmodic action."[3]

Donnagel® treats the whole diarrhea problem.

Available in new 4-ounce plastic bottle on your prescription or recommendation. Also available: Donnagel®-PG (with paregoric equivalent) and Donnagel® with Neomycin. See product literature before prescribing.

References: 1. Kramer, P., and Ingelfinger, F.J.: Med. Clin. N. Amer., *32*:1227, 1948. 2. Hock, C.W.: Clin. Med., *8*:1932, 1961. 3. Winfield, I.W.: Am. J. Gastroent., *31*:438, 1959.

A-H-ROBINS

A. H. Robins Company, Inc.
Richmond, Virginia 23220



"CLEAR THE TRACT" WITH

Coughing ahead...
Clear the Respiratory Tract with Robitussin.

Much more than just a slogan, "clear the tract" reflects the dependable antitussive-expectorant action of the three Robitussin formulations. All contain glyceryl guaiacolate, the time-tested expectorant that greatly enhances the output of lower respiratory tract fluid. Increased RTF volume exerts a demulcent effect on the tracheobronchial mucosa, promotes ciliary action, and makes thick, inspissated mucus less viscid and easier to raise. Glyceryl guaiacolate is safe, non-narcotic, and almost universally accepted by patients of all ages.

NOW! THREE ROBITUSSIN FORMULATIONS	ROBITUSSIN	ROBITUSSIN A-C	ROBITUSSIN-DM
EXPECTORANT	●	●	●
DEMULCENT	●	●	●
COUGH SUPPRESSANT		●	●
ANTIHISTAMINE		●	
LONG-ACTING (6-8 hours)			●

FORMULAS

ROBITUSSIN®
in each 5 cc. teaspoonful:
Glyceryl guaiacolate 100 mg.
(Alcohol 3.5%)

ROBITUSSIN® A-C
(exempt narcotic)
in each 5 cc. teaspoonful:
Glyceryl guaiacolate 100 mg.
Pheniramine maleate 7.5 mg.
Codeine phosphate 10.0 mg.
(warning: may be habit forming)
(Alcohol 3.5%)

ROBITUSSIN®-DM
new, non-narcotic
in each 5 cc. teaspoonful:
Glyceryl guaiacolate 100 mg.
Dextromethorphan hydrobromide 15 mg.

Robitussin and Robitussin-DM are available at pharmacies everywhere on your prescription or recommendation.

A. H. Robins Company, Inc. Richmond, Va.

OUR PHOTO:
Engine No. 89 of the Monadnock, Steamtown & Northern Railway pulls a trainload of steam enthusiasts through the New England countryside between Bellows Falls and Chester, Vermont.

A·H·ROBINS

ONE OF THE ROBITUSSIN® FORMULAS

vignettes of angina pectoris —
no. 1 in a series:

angina and the surgeon

John Hunter —
British surgeon (1728-1793)

angina of anger

"My life is in the hands of any rascal who chooses to annoy and tease me."[1] So said the great British surgeon and anatomist, John Hunter, realizing that he could not control the anger which precipitated frequent and severe attacks of angina pectoris. According to Mettler: "His statement was no exaggeration. On October 16, 1793, he attended a meeting of the St. George's hospital staff, and, while defending the interests of several students, he was contradicted and thoroughly antagonized. The pains of angina commenced, he started toward another room, gained it, and fell dying into the arms of a physician."[2]

Why Edward Jenner withheld his paper on angina In 1777, at an earlier stage of the condition, Hunter's angina alarmed a favorite pupil, Edward Jenner, who wrote to Dr. Heberden that he feared his teacher was "affected with symptoms of the Angina Pectoris."[3] So concerned was Jenner about his former teacher's emotion-related condition that he deliberately cancelled publication of a paper on angina pectoris, fearing that Hunter would read it, and have "his fears excited by its truly formidable nature."[4]

Severity of angina described Hunter's brother-in-law, Dr. Everard Home, who witnessed his death and performed an autopsy, gave this account of the later stages of the condition:

"... the pain became excruciating at the apex of the heart; the throat was so sore as not to allow of an attempt to swallow anything and the left arm could not bear to be touched....

"The affections above described were, in the beginning, readily brought on by exercise ... but they at last seized him when lying in bed, and in his sleep...."[5]



18th century ancestor of the modern coronary candidate Surgeon, anatomist, pathologist, physiologist, geologist, and teacher Hunter had a passion for research which led him to disregard his practice, his health and even the

law. When the Irish giant O'Brien learned that Hunter desired his skeleton for a museum, he willed that his body be sunk at sea in a lead coffin. But Hunter was not to be denied. According to Major, he "... bribed the watchers and finally obtained the body at a cost of 500 pounds although he had to borrow the money to pay the men."[3] In 1767, he experimentally inoculated himself with gonorrhea and syphilis, treated himself with mercury for three years, and was apparently cured.[5] Hunter had feelings of inadequacy about his education and speaking ability, but this did not prevent him from being hard driving and abrupt with his colleagues. His competitiveness with his physician older brother was also well known, and ended in complete estrangement between the two men.[2,3] Today, the personality traits seen in John Hunter are recognized to be important predisposing factors in the development of coronary artery disease—often manifested as angina pectoris. According to Friedman and Rosenman,[6] in a group of men whose behavior was characterized by intense ambition and competitive drive, a greater than average incidence of angina pectoris was among those abnormal conditions noted.

References: 1. Paget, S., cited by Mettler, C. A.: History of Medicine, Philadelphia, The Blakiston Company, 1947, p. 85. 2. Mettler, C. A.: *Op. cit.*, pp. 84-85. 3. Major, R. H.: A History of Medicine, Springfield, Ill., Charles C Thomas, 1954, vol. 2. pp. 601-607. 4. Baron, J., cited by Major, R. H.: *Op. cit.*, p. 607. 5. Major, R. H.: Classic Descriptions of Disease, ed. 3, Springfield, Ill., Charles C Thomas, 1955, p. 423. 6. Friedman, M., and Rosenman, R. H.: J.A.M.A. *169*:1286, 1959.

in the modern management of angina pectoris

Peritrate® SA

Sustained Action (pentaerythritol tetranitrate) 80 mg.

Each double-layer, biconvex, dark green/light green tablet of Peritrate SA Sustained Action contains:
pentaerythritol tetranitrate ..... 80 mg.
(20 mg. in immediate release layer and 60 mg. in sustained release base)

Peritrate (pentaerythritol tetranitrate) is a nitric acid ester of a tetrahydric alcohol (pentaerythritol).

Actions: The exact cause of angina pectoris (that is, the pain associated with coronary artery disease) remains obscure, despite the numerous and often conflicting hypotheses concerning its pathophysiology. Therapy at the present time, therefore, remains essentially empiric. Customarily, clinical improvement has been measured by: reduction in (1) number, intensity and duration of angina pectoris attacks and (2) necessity for glyceryl trinitrate intake for prevention or relief of anginal attacks.

Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. has been reported in clinical usage to reduce in number and severity the incidence of angina pectoris attacks, with concomitant reduction in glyceryl trinitrate intake.

In the evaluation of Peritrate (pentaerythritol tetranitrate) and Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. in angina pectoris, clinical improvement has been customarily measured subjectively by reduction in number and severity of attacks and necessity for glyceryl trinitrate intake for prevention or abortion of anginal attacks. Individual patterns of angina pectoris differ widely as does the symptomatic response to antianginal agents such as pentaerythritol tetranitrate. The published literature contains both favorable and unfavorable clinical reports. In conjunction with total management of the patient with angina pectoris, Peritrate (pentaerythritol tetranitrate) and Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. have been accepted as safe for prolonged administration and widely regarded as useful.

Animal pharmacology: In a series of carefully designed studies in pigs, Peritrate (pentaerythritol tetranitrate) was administered for 48 hours before an artificially induced occlusion of a major artery and for seven days thereafter. The pigs were sacrificed at various intervals for periods up to six weeks. The result showed a significantly larger number of survivors in the drug-treated group. Damage to myocardial tissue in the drug-treated survivors was less extensive than in the untreated group. Pigs rather than dogs were used because their coronary artery distribution more closely resembles that of human beings. Studies in dogs subject to oligemic shock through progressive bleeding have demonstrated that Peritrate (pentaerythritol tetranitrate) is vasoactive at the postarteriolar level, producing increased blood flow and better tissue perfusion. These animal experiments cannot be translated to human behavior

Indications: Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. is indicated for the relief of angina pectoris (pain associated with coronary artery disease). It is not intended to abort the acute anginal episode but is widely regarded as useful in the prophylactic treatment of angina pectoris.

Contraindications: Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg. is contraindicated in patients who have a history of sensitivity to the drug.

Warning: Data supporting the use of Peritrate (pentaerythritol tetranitrate) during the early days of the acute phase of myocardial infarction (the period during which clinical and laboratory findings are unstable) are insufficient to establish safety.

Precautions: Should be used with caution in patients who have glaucoma.

Adverse reactions: Side effects reported to date have been predominantly related to headache (which may require discontinuation of medication) and gastrointestinal distress which are usually transient with continuation of medication.

Dosage: Peritrate SA Sustained Action (pentaerythritol tetranitrate) 80 mg., 1 tablet immediately on arising and 1 tablet 12 hours later (on an empty stomach).

Additional dosage forms
Peritrate (pentaerythritol tetranitrate)—10 mg. and 20 mg. tablets with or without phenobarbital.

Peritrate with Phenobarbital SA Sustained Action—80 mg. pentaerythritol tetranitrate and 45 mg. phenobarbital.

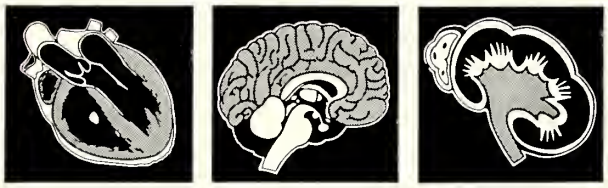
(**Warning:** Tablets containing phenobarbital may be habit forming.)

WARNER-CHILCOTT
Morris Plains, N. J.

Behind continued high blood pressure readings lies the possibility of organic damage[1-13]

MANY OF THE aspects of essential hypertension are unpredictable—either because there are a number of mechanisms involved or because individuals differ in their responses to these mechanisms.[1]

There is one aspect of hypertension, however, that seems, in many cases, predictable. ". . . when the blood pressure is elevated to a marked degree for an adequate period of time, this in itself leads to perpetuation of the syndrome with resulting vascular damage throughout the body."[14] All too often the disease progresses until there is damage to one of three vital organs: the heart, the kidney, the brain.



"Hypertension is certainly a major factor in the genesis of coronary heart disease, and it is even more important when compounded with obesity."[4]

"[Vascular deterioration] can be clearly seen in the kidney with a degree of damage that can be measured by renal function studies."[10]

". . . most evidence suggests that reduction of blood pressure, when it is too high, not only relieves the heart of excess work but reduces vascular damage."[1]

"In short, treatment is indicated."[1]

Antihypertensive therapy will not restore the blood vessels to normal. Yet many of the vascular changes and symptoms caused by increased blood pressure may be arrested or alleviated when the blood pressure is reduced to normotensive levels.[7]

Reducing the blood pressure helps curtail further vascular damage and improves the prognosis — when damage is not too far advanced before therapy is started.[14] Essential hypertension is an indication not only for treatment, but for *early* and adequate treatment of the patient in question.

Reduce the blood pressure with Rautrax-N

Rautrax-N combines the antihypertensive-tranquilizing action of whole root rauwolfia with the antihypertensive-diuretic action of bendroflumethiazide in one convenient medication. The two drugs complement each other so that smaller doses of both are possible.

Rauwolfia combined with bendroflumethiazide is particularly effective in long-term therapy,[15-17] since beneficial effects do not diminish with continuous daily administration.

For most patients 1 or 2 Rautrax-N tablets daily are sufficient for maintenance therapy. The simplicity, convenience and economy of such a dosage schedule are of particular benefit to older patients.

References: 1. Page, I. H., and Dustan, H. P.: The Usefulness of Drugs in the Treatment of Hypertension, in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 95. **2.** Hollander, W.: The Evaluation of Antihypertensive Therapy of Essential Hypertension in Ingelfinger, F. J.; Relman, A. S., and Finland, M.: Controversy in Internal Medicine, Philadelphia, W. B. Saunders Co., 1966, p. 97. **3.** Nickerson, M.: Antihypertensive Agents and the Drug Therapy of Hypertension, in Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 3, New York, The Macmillan Co., 1965, p. 727. **4.** Berkson, D. M.: Indust. Med. & Surg. **32**:371, 1963. **5.** Cohen, B. M.: M. Times **91**:645, 1963. **6.** Lee, R. E., et al.: Am. J. Cardiol. **11**:738, 1963. **7.** Moyer, J. H.: Am. J. Cardiol. **9**:821, 1962. **8.** Moser, M.: New York J. Med. **62**:1177, 1962. **9.** Wood, J. E., and Battey, L. L.: Am. J. Cardiol. **9**:675, 1962. **10.** Moyer, J. H., and Heider, C.: Am. J. Cardiol. **9**:920, 1962. **11.** Moser, M., and Macaulay, A. I.: New York State J. Med. **60**:2679, 1960. **12.** Judson, W. E.: Nebraska M. J. **44**:305, 1959. **13.** Hodge, J. V.; McQueen, E. G., and Smirk, H.: Brit. M. J. **1**:5218, 1961. **14.** Moyer, J. H., and Brest, A. N.: Hypertension Recent Advances, Philadelphia, Lea & Febiger, 1961, p. 633. **15.** Berry, R. L., and Bray, H. P.: J. Am. Geriatrics Soc. **10**:516, 1962. **16.** Reid, W. J.: J. Am. Geriatrics Soc. **13**:365, 1965. **17.** Feldman, L. H.: North Carolina M. J. **23**:248, 1962.

Contraindications: Severe renal impairment or previous hypersensitivity. **Warning:** Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting or G.I. bleeding occur.

Precautions and Side Effects: The dose of ganglionic blocking agents, veratrum or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Caution is indicated in patients with depression, suicidal tendencies, peptic ulcer; electrolyte disturbances are possible in cirrhotic or digitalized patients. Marked hypotension during surgery is possible; consider discontinuing two weeks prior to elective surgery and observe patients closely during emergency surgery. Rauwolfia preparations may cause reversible extrapyramidal symptoms and emotional depression, diarrhea, weight gain, edema, drowsiness may occur. Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients, and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, rashes may occur.

Dosage and Supply: Initial dosage, 1 to 4 tablets daily, preferably at mealtime. Maintenance, 1 or 2 tablets daily. Rautrax-N is supplied as capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin®), 4 mg. bendroflumethiazide (Naturetin®), 400 mg. potassium chloride.

Also available: Rautrax-N Modified — capsule-shaped tablets containing 50 mg. Rauwolfia serpentina whole root (Raudixin), 2 mg. bendroflumethiazide (Naturetin), 400 mg. potassium chloride. Both potencies available in bottles of 100. For full information, see Product Brief.

RAUTRAX®-N

Squibb Rauwolfia Serpentina Whole Root (50 mg.) with Bendroflumethiazide (4 mg.) and Potassium Chloride (400 mg.)

SQUIBB 'The Priceless Ingredient' of every product is the honor and integrity of its maker.



And now . . . for all you cold sufferers who've been looking for a cure-all.

They can't cure a cold. We can't cure a cold. You can't cure a cold. But what you can do is relieve the symptoms, making the patient comfortable and the cold bearable.

The patient suffering from head cold congestion, for instance, should breathe easier when you prescribe Novahistine LP.

Novahistine LP is a long-acting decongestant that helps restore normal mucus secretion and ciliary activity—physiologic mechanisms which prevent infection of the respiratory tract. Two tablets in the morning and two in the evening will provide around-the-clock relief by helping to keep congested air passages clear, thus enabling your cold patient to enjoy normal and free breathing.

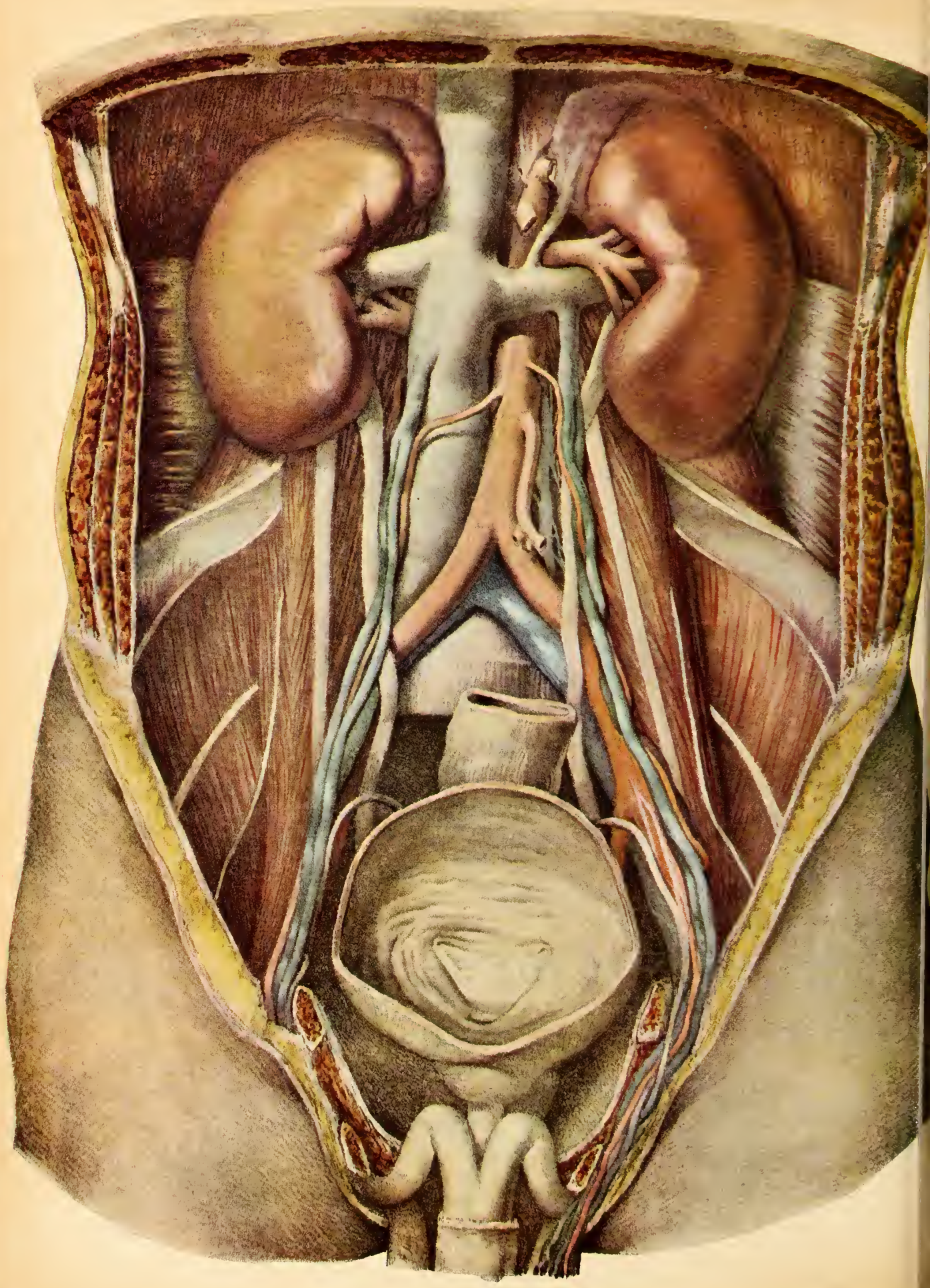
Use cautiously in individuals with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Tell patients who operate machinery or motor vehicles that drowsiness may result.

Each Novahistine LP tablet contains: phenylephrine hydrochloride, 25 mg., and chlorpheniramine maleate, 4 mg.

PITMAN-MOORE Division of The Dow Chemical Company, Indianapolis DOW

NOVAHISTINE® LP

For relief of nasal congestion.



Wide-range bactericidal action for genitourinary infections

NEW OMNIPEN® (AMPICILLIN) WYETH

A penicillin that exhibits effectiveness within the gram-positive spectrum of penicillin G* and the gram-negative spectrum of chloramphenicol and the tetracyclines.*

Active at foci of infections—kidney, ureter, bladder or urethra.

Effective against many gram-negative and gram-positive pathogens—thus may be valuable not only in genitourinary but also in common respiratory and gastrointestinal infections.

Normally produces high and persistent levels in blood and high concentrations in bile and urine.

Significant inherent stability.

**Exclusive of penicillinase-producing bacteria.*

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A Critique of the Durham Rule

(Maine's Modified Durham Rule)

PETER W. BOWMAN, M.D.* and COURTLAND D. PERRY, Esquire**

Society's awareness that a crime can be the result of mental illness or of defective intelligence, as acknowledged and reflected in the criminal law, is relatively new.

In the leading case of *State vs. Lawrence*,[1] 1870, Maine first adopted the "right-wrong" test of criminal responsibility as first enunciated in Daniel M'Naghten's case,[2] citing with approval the trial court's instruction to the jury as follows:

"To excuse a man from responsibility on the ground of insanity, it must appear, that at the time of doing the act he had no capacity and reason sufficient to enable him to distinguish between right and wrong, as to the particular act he was doing. That he had not knowledge, consciousness, or conscience enough to know, that the act he is doing is a wrong act, and a criminal act, and one that he will be subject or liable to punishment for doing. In order to be responsible, he must have sufficient mind and memory to understand and remember the relation he stands to others, and others to him, and that the act he is doing is contrary to the plain dictates of right, wrongfully injurious to others, and a violation of the dictates of duty. It there be partial derangement, or insanity of mind, so that it is not in all respects perfectly sane and sound, yet if not to the extent above indicated, it will not excuse a criminal act. In other words, a man may be a monomaniac, his mind may be disordered, and, to a certain extent, it may be proved that he is of unsound mind, and yet, if he has mind and understanding enough, and is not carried away so but that he understands the difference between right and wrong, as to the act he is then doing, – that is to say, if the man knew that what he was doing was wrong, and he was liable to be punished for it, and that the act would not be excused, then he is subject to punishment, although there might be some partial derangement."

The "right-wrong" test controlled the jury's disposition of the insanity plea in Maine until 1961 when the 100th Maine Legislature leaving a Legislative Record barren of debate enacted the "Durham Rule,[3]" which with legislative modification appears as follows:

"An accused is not criminally responsible if his unlawful act was the product of mental disease or mental defect. The terms 'mental disease' or 'mental defect' do not include an abnormality manifested only by repeated criminal conduct or excessive use of drugs or alcohol."[4]

In establishing a frame of reference for our discussion of insanity and the criminal law in Maine we direct our attention to two decisions of our Maine Supreme Judicial Court, interpreting Maine's new test of criminal responsibility following enactment of Maine's modified "Durham Rule" in 1961. After enactment there was some discussion among lawyers as to whether the statute actually superseded *M'Naghten*. The Court in *State of Maine vs. Park*[5] removed any question by stating:

"The line between criminal responsibility and lack of criminal responsibility is drawn by our 1961 statute. We then discarded the McNaughten Rule and adopted the Durham Rule with modifications."

The Court in *Park* rejected the doctrine of partial responsibility, a construction attempted to be given the new test by the defendant:

"We had no such rule of partial or limited responsibility under the McNaghten Rule. There is nothing inherent in our Durham Rule requiring the creation of a new zone of uncertain width with changing shadows for the benefit of those charged with crime."[6]

The Court defined the function of the jury and declined to follow *Lyles vs. U.S.*,[7] and other cases which held that the defendant, unless he waived it, was entitled to have the jury instructed as to the consequences of an acquittal by reason of insanity:

"It has long been the settled practice in our State that the function of the jury is to find the facts and to apply the law as given by the court to the facts in reaching their verdict. Punishment, or whatever may transpire after the verdict, is not the concern of the jury."[8]

To complete the Court's circumscription of practice

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under the modified "Durham Rule" it reiterated the holding in *State vs. Lawrence* as to the question of burden of proof, by acquiescing in the trial court's instruction to the jury, that the burden is on the defendant to prove insanity by a fair preponderance of the evidence, an evidentiary rule in our view, more functional, and a burden more easily met, than that which obtains in the District of Columbia.

In *State of Maine vs. Hathaway,*[9] the Court adopted the holding of *McDonald vs. United States,*[10] and finding reversible error in the trial court's instruction to the jury on the question of the scope of the test to be applied said:

". . . Such instructions constituted a reversion to the restrictive and superseded M'Naghten Rule of State v. Lawrence, 57 Maine at 576. The instructions rendered deprived the defendant of jury consideration and estimation of the medical testimony presented upon the grave issue of defendant's inability because of some mental disease or defect to refrain from the imputed homicide irrespective of defendant's knowledge of right and wrong at the time of the tragic incident. The charge was therefore elliptical and insufficient. The jury were not informed that they could and must decide whether upon all the evidence the defendant had proved by a preponderance that at the occurrence of the fatal event the defendant's mental or emotional processes were substantially affected and his behavior controls were substantially impaired."

Thus, for good or for bad, as we shall later discuss, the transition from the "right-wrong" test to the "product test" has been made, the only State legislative enactment of its kind; as Maine has gone twenty-two other States have declined to go.[11]

The rule regarding criminal responsibility has changed; however, the consequences to the defendant who prevails today are as untenable as they were at the time of State v. Lawrence, as seen from an examination of the following laws:

R.S., 1871, c. 137, Secs. 2,3.

Sec. 2. "When the grand jury omits to find an indictment against any person arrested by legal process to answer for any offence, by reason of his insanity, they shall certify that fact to the court; and when a traverse jury, for the same reason, acquits any person indicted, they shall state that fact to the court when they return their verdict; *and the court, by a precept stating the fact of insanity, may commit him to prison or to the insane hospital, till restored to his right mind or delivered according to law; but he shall only remain in prison till provision can be made for him at the hospital, and then removed thereto."*
(Emphasis Ours)

Sec. 3. *"Any person so committed may be discharged by any judge of the supreme judicial court, in term time or vacation, on satisfactory proof that his discharge will not endanger the peace and safety of the community;* or the judge may, on application, commit him to the custody of any friend, who gives bond to the judge of probate for the County of Kennebec, with sufficient sureties, approved by said judge of probate, conditioned for the safe keeping of such insane person, and the payment of all damages which any person may sustain by his acts. And when, on satisfactory proof, he is again found insane, and dangerous, any judge of the supreme judicial court may, by a precept stating the fact of his insanity, recommit him to the insane hospital." (Emphasis Ours)

T., 15, M.R.S.A. 1964, Secs. 103, 104

Sec. 103. "When a respondent is acquitted by reason of mental disease or mental defect excluding responsibility, the verdict and judgment shall so state. In such case the court shall order such person committed to the custody of the Commissioner of Mental Health and Corrections to be placed in an appropriate institution for the mentally ill or the mentally retarded for care and treatment. . . ." (Emphasis Ours)

T., 15, M.R.S.A. 1964, Sec. 104

Sec. 104. "Upon a petition to the Superior Court in the county where committed or to a justice, in vacation, in said county, *and upon satisfactory proof that his discharge, or release in the custody of a relative or friend, will not endanger the peace and safety of the public,* said court or justice may discharge, or release in the custody of a relative or friend, the person committed under section 103. . . . When released in the custody of a relative or friend such relative or friend shall give bond in an amount to be determined by said court or justice, to the judge of probate for the county from which committed, with sufficient sureties, approved by said judge of probate, conditioned for the safekeeping of such person, and the payment of all damages which any person may sustain by his acts, occasioned by his mental disease or mental defect . . ."

At the time of *State vs. Lawrence,* it was mandatory that the Court commit the defendant acquitted by reason of insanity and even now under our modified "Durham Rule," mandatory commitment follows acquittal on the ground of "mental disease" or "mental defect" excluding responsibility, resulting in *indeterminate* confinement, release from which is dependent upon proof of absence of dangerousness.

If we may ascribe to the Maine Legislature knowledge and acceptance of the intended purpose of the *Durham* decision we may say that the enactment of Maine's modified "Durham Rule" was intended to broaden the scope of psychiatric testimony in criminal cases from statements regarding the defendant's capacity to understand the criminality of his act, to testimony encompassing the full range of current psychiatric knowledge pertinent to the case at trial. We may further say that its purpose was to exculpate the accused, who, although he understood the criminality of his act, committed the act due to mental abnormality, i.e., the irresponsible should not be punished. We then ask: "Has the new rule fulfilled its intended purpose and what in practice is its effect?"

Maine's "Durham Rule" contains a broad and generalized reference to mental disease and mental defect and has a number of psychiatric presuppositions which one of the authors[12] discussed in a previous paper as follows:

1. *Mental Disease:* The law implies that mental disease is a well-defined and generally accepted concept; that it has clear-cut lines of demarcation toward mental health; that it is a disease process which can either improve or deteriorate; that it has a demonstrable pathologic substrate.

2. *Mental Defect:* The law presupposes that mental defect is a clear-cut entity; that it has been defined acceptably; that it can be measured accurately.

When one tests the reality of these presuppositions with respect to mental disease, one has to make some disconcerting observations:

The Diagnostic and Statistical Manual, Mental Dis-

orders, American Psychiatric Association, 1952, on Page 1, refers to "diseases of the psychobiologic unit" after which specific conditions, presumably diseases, are listed as "disorders." Thus, "disease" and "disorder" are used interchangeably.

Webster's dictionary defines "disease" as: A condition in which bodily health is impaired; sickness; illness; also, a malady; an ailment. The same source defines "disorder" as: 1.) Lack of order; confusion; disarray. 2.) Neglect of order or system; irregularity. 3.) Breach of public order; tumult. 4.) Sickness; ailment. v.t. 1.) To disarrange; to confuse. 2.) To disturb the natural functions of (body or mind); to derange.

We doubt that there is agreement between psychiatrists or even physicians as to what constitutes a "disorder" and what is a "disease," although this is conceivable. I do not think, however, that any physician will use "disease" and "disorder" interchangeably, implying a synonymous definition.

Since the psychiatrist will find himself in the legal halls of society, when involved in criminal procedures, it is interesting to find out how the law defines "disease" and "disorder":

Black's Law Dictionary, 4th Ed., 1951, defines "disease" as deviation from the healthy or normal condition of any of the functions or tissues of the body; an alteration in the state of the body or of some of its organs interrupting or disturbing the performance of the vital functions and causing or threatening pain and sickness; illness; sickness; disorder; malady; bodily infirmity.

The same source defines "disorder" as: turbulent or riotous behavior; immoral or indecent conduct; the breach of the public decorum and morality. 2. Is usually a slight, partial, and temporary physical ailment.

Turning back to the Statistical Manual of the American Psychiatric Association, one notes that a group of disorders are classified according to symptoms with attempted etiological identification under "acute brain disorders" and "chronic brain disorders," both having demonstrable organic pathology.

The psychotic disorders come under the heading of "disorders of psychogenic origin or without clearly defined cause or structural change in the brain." They include manicdepressive, schizophrenic, and paranoid reactions.

Under the same heading, we note the psychosomatic, psychoneurotic, and the personality disorders.

Thus, a major psychosis such as schizophrenia, a psychoneurotic anxiety reaction, a psychosomatic skin reaction, and a sociopathic personality disturbance, are all considered "disorders of psychogenic origin or without clearly defined cause or structural change in the brain."

Does this imply that the patient with psoriasis, the patient with an acute or chronic anxiety or phobic reaction, is mentally ill and should be excused if found guilty of a criminal offense?

Does this imply that the anti-social actions of the criminal is a product of mental disease?

The intent of the law surely does not favor such approach. In its present wording, however, it would leave the door wide open to an abuse of provisions that were meant to protect certain mentally deranged people against inappropriate punishment. Therefore, it had been suggested that an Interdisciplinary Committee, composed of members of the Maine Medical Association and the Maine Bar Association, study the possibility of replacing the words "mental disease" by specific identifiable pathologic disease entities which should be included in the law.

In reference to "mental defect" the confusion is equally bewildering.

The Statistical Manual of the American Psychiatric Association refers to this item as including "disorders due to unknown or uncertain cause with the functional reaction alone manifest; hereditary and familial diseases of this nature."

(Note, again, the interchangeable use of "disorder" and "disease".)

Sub-classifications into degrees of "mental defect" are offered and to be arrived at by testing. However, the test or tests to be used are not identified.

This section reflects a degree of ignorance on the part of the profession that is not in keeping with our actual knowledge in 1965.

The Group for the Advancement of Psychiatry in its Report No. 43,[13] in 1959, pointed out that the terms "mental retardation," "mental deficiency," "feebleminded," "mental inadequacy," and "sub-normality," refer to a broad phenomenon indicating unusually slow or arrested development and decided to use the term "mental retardation," a national trend not yet reflected in the American Psychiatric Association Manual.

The Group for the Advancement of Psychiatry further acknowledges that "modern advances in the medical sciences have made us increasingly aware of the multiplicity of the factors in the etiology of mental retardation" and submits that known etiological factors "will fall into one of the following categories: somatic, cultural, social, psychological."

At any rate, it is our experience that some individuals with impaired learning ability, functioning in the borderline range of intelligence, or in the mild range of retardation, actually have concurrent personality trait disturbances that are unrelated to intellectual functioning.

If convicted of criminal offenses, these individuals are properly placed in correctional institutions regardless of the fact that their intelligence quotient may measure only 65 or 70. The rare occasion in this category is the patient who simultaneously suffers from an epileptic syndrome or a metabolic disturbance which probably should be covered under the section of specific diseases, replacing the words "mental disease," as proposed.

Patients with a mental retardation syndrome, whose disability places them in the trainable or dependent range of intellectual deficit are usually considered in-

capable of premeditated crimes and if found guilty of such a crime, it was most likely either the result of an accident or it was instigated and supervised by a competent person.

In these cases, it is rare if, indeed, ever necessary, to place such a person under security provisions. Adequate social supervision as offered in institutional or group living as offered in nursing homes, sheltered workshops, etc., will suffice.

For didactic purposes, we would like to summarize now that we have no valid or commonly accepted definitions of either "mental disease" or of "mental defect" yet these 2 terms are part of a law that presupposes that such definitions do exist.

Next, we wish to explore criteria used by psychiatrists when testifying as expert witnesses.

The testimony given, at least in the majority of the cases, is based upon examinations that took place after the crime was committed. However, the psychiatrist is asked whether the respondent's crime, if, indeed, he committed one, was the result of "mental disease" or "mental defect."

Can a qualified psychiatrist, beyond any reasonable doubt, testify *in every case* to a condition of which he had no knowledge at the time of the alleged crime? Surely, there are many cases where this can be done. However, there are others where this may be difficult or even impossible.

In other words, the psychiatrist may be asked to testify to a "mental disease" that he did not witness at the time it was supposed to have existed and he may further testify that the crime committed was "the result of mental disease" even though the jury has not even found that the crime was committed by the respondent whose "mental disease" the psychiatrist may be asked to testify has caused the crime.

One wonders whether psychiatrists have this capability. One rather thinks not.

However, upon legal interpretation, we are drawn to the conclusion that the statute does not presuppose precise definitions of "mental disease" and "mental defect." It *does* presuppose psychiatry to be a dynamic discipline and presupposes that the psychiatrist's role is to impart to the jury, based on his knowledge of his medical specialty, his opinions regarding the existence of what he considers to be a mental disease or a mental defect, leaving to the jury the ultimate question, the actual determination of the existence of the disease or defect.

If we can assume for the moment that the psychiatrist's role with respect to criminal proceedings is desirable under *Durham,* certainly his free reign in the courtroom would be destroyed by legislative definition of "pathologic disease entities"; the flexibility of the rule would be lost. Psychiatry, now recognized by the rule to be dynamic, would be rendered static by such restriction of definitions. As knowledge in the field grows, under such restrictions we would be forced legislative session after session to amend the definitions. Furthermore, although the "pathologic disease entities" would be defined, the psychiatrists would still disagree as to the manifested symptomatology necessary to comprise such entities, and the jury would still be confused.

This discussion, however, points out one of the basic weaknesses of *Durham* in practice; that is, the failure or inability of the psychiatrists to agree as to the nature of mental disease or mental defect; placing the disagreeing experts in the adversary trial climate, and leaving to the ill-equipped jury the problem of resolving the question upon which the experts disagree.

One State psychiatrist, who has testified in more than one hundred criminal cases, under *M'Naghten* and under *Durham,* states that his role differs little under the new rule as compared with his role under the old, indicating to the writers that the Maine trial courts allowed the psychiatrist a fairly broad scope of testimony under the old rule.[14]

Under the *Hathaway* decision, lay testimony is equated to psychiatric testimony resulting in an adulteration of the purpose of *Durham,* inasmuch as the jury on the question of mental responsibility must not only weigh carefully unknown psychiatric terms and concepts, but must weigh them diluted by the testimony of lay witnesses.

We are, indeed, concerned with the role of the psychiatrist with respect to the "product" aspect of the rule, but also, or even more so, with the task of the jury in this regard and *it is the ultimate question for the jury.* The element of causation is, indeed, difficult and seemingly impossible for the jury to determine because its necessary relationship to the "disease" or "defect" to justify acquittal is yet undetermined.

In considering the shortcomings of the modified "Durham Rule," we should examine closely the modification itself and ask the question: Does an "abnormality," manifested only by repeated criminal conduct or excessive use of drugs or alcohol, exist? We think not. By the exclusion, the "sociopath," so-called, is purportedly removed from the scope of the terms "mental disease" or "mental defect." We would contend that repeated criminal conduct and excessive use of drugs or alcohol cannot alone be the manifestation of "abnormality" but that some other aberration exists in every case capable of diagnostic identification.

Further, we have stated that the "Durham Rule" intended to relieve from punishment – the irresponsible, yet we find that the defendant who prevails, who is acquitted by reason of "mental disease" or "mental defect" excluding responsibility is automatically committed to the custody of the Commissioner of Mental Health and Corrections to be placed in an institution for the mentally ill or mentally retarded for care and treatment, and he may not resume his place in the community until the Court can be satisfied that his release or discharge will not endanger the peace and safety of the public. Thus, although he may be cured of his illness *he may spend his life in a State institution.* He is purportedly civilly

committed for care and treatment and yet criteria of criminality are applied in determining his fitness for release or discharge without reference to his mental condition.

Summary

The effect of the modified "Durham Rule" we find, in summary, to be as follows:

The jury are asked to shoulder the burden of determining the existence of "mental disease" or "mental defect," frequently from conflicting psychiatric testimony loaded, as it were, with terminology and professional opinions with which the experts disagree, and are charged with the impossible task of determining the causative relationship between the "mental disease" or "mental defect," and the criminal act, without an understanding or guide as to the necessary relationship between the two, to justify acquittal.

We find that the jury cannot consider the consequences of acquittal, i.e., indeterminate confinement, nor should they, but we also disconcertedly find that no one else has considered it. The defendant who committed a crime against property, which, if convicted, would have resulted in a relatively short sentence with parole and its supervision and guidance upon return to the community, if acquitted by reason of "mental disease" or "mental defect" excluding responsibility is subjected to a possible lifetime of confinement in a State institution.

And what of the role of the psychiatrist who must attempt to diagnose a "mental disease" weeks and sometimes months after the time at which its existence is pertinent and the act to which it is to be related took place? What can be done to cast the psychiatrist, the judge and the jury, in their proper roles and to assure the appropriate disposition of criminal cases in the interest of the defendant and of the public? The jury should, perhaps, be relieved of its awesome burden imposed by *Durham* and should be assigned a task commensurate with their traditional role of exercising the judgment of laymen on issues and concepts, within their understanding, i.e., the return to *M'Naghten.*[15] Further, the law should be amended to make commitment after acquittal discretionary with the Court.

The psychiatrist, besides his testimony under the limited *M'Naghten* test which will result in the exculpation of the severely psychotic and retarded, we suggest should be permitted to exercise the full range of his professional knowledge, supported by the findings of experts in related disciplines, at the time of sentence, apart from the adversary climate of the trial.

Such proposal is not entirely new in Maine. For several years, Interim Legislative Committees and Administrative Personnel of the Department of Mental Health and Corrections have studied the need for, and feasibility of, a reception and treatment center, staffed by qualified professionals in psychiatry, psychology, sociology, etc., the function of which it has been contemplated would be to receive the sentenced offender and determine the appropriate placement for him, considering his needs and those of the public. The center would have an adequate treatment unit designed to administer to the needs of the "sociopath," the offender rejected by the Maine State Prison and by the mental hospitals. Sentencing procedures would have to be altered under this system.

We would suggest *the reversal of this process,* i.e., the use of the proposed reception center as a diagnostic center to which certain offenders would be sent for a complete diagnostic workup, the report of which, with psychiatric testimony, would be presented to the court for its advice and guidance as to the appropriate sentence, and would recommend the Model Sentencing Act, or something like it, as the vehicle for implementing the proposed system. The pertinent text of the Act follows:

Sec. 5. Dangerous Offenders

"Except for the crime of murder in the first degree, the court may sentence a defendant convicted of a felony to a term of commitment of thirty years, or to a lesser term, if it finds that because of the dangerousness of the defendant, such period of confined correctional treatment or custody is required for the protection of the public, and if further finds, as provided in section 6, that one or more of the following exist:

"(a) The defendant is being sentenced for a felony in which he inflicted or attempted to inflict serious bodily harm, and the court finds that he is suffering from a severe personality disorder indicating a propensity toward criminal activity. (b) The defendant is being sentenced for a crime which seriously endangered the life or safety of another, has been previously convicted of one or more felonies not related to the instant crime as a single criminal episode, and the court finds that he is suffering from a severe personality disorder indicating a propensity toward criminal activity. (c) The defendant is being sentenced for the crime of extortion, compulsory prostitution, selling or knowingly and unlawfully transporting narcotics, or other felony, committed as part of a continuing criminal activity in concern with one or more persons.

The findings required in this section shall be incorporated in the record.

Sec. 6. Procedure and Findings

"The defendant shall not be sentenced under subdivision (a) or (b) of section 5 unless he is remanded by the judge before sentence to (diagnostic facility) for study and report as to whether he is suffering from a severe personality disorder indicating a propensity toward criminal activity; and the judge, after considering the presentence investigation, the report of the diagnostic facility, and the evidence in the case or on the hearing on the sentence, finds that the defendant comes within the purview of subdivision (a) or (b) of section 5. The defendant shall be remanded to a diagnostic facility whenever, in the opinion of the court, there is reason to believe he falls within the category of subdivision (a) or (b) of section 5. Such remand shall not exceed ninety days, subject to additional extensions not exceeding ninety days on order of the court.

"The defendant shall not be sentenced under subdivision (c) of section 5 unless the judge finds, on the basis of the presentence investigation or the evidence in the case or on the hearing on the sentence that the defendant comes within the purview of the subdivision. In support of such findings it may be shown that the defendant has had in his own name or under his control substantial income or resources not explained to the satisfaction of the court as derived from lawful activities or interests."[17]

Under the above proposals, the severely psychotic and retarded, would be exculpated, and the offender who

knew and understood the criminality of his act would be sentenced to a therapeutically oriented correctional system prepared to serve his needs and those of the community. Such a system would remove the punishment versus treatment argument, and would permit the psychiatrist as to cases now exculpated under *Durham,* to *prescribe treatment,* to advise the court as to the total needs of the defendant, instead of trying to inform the jury, who are incapable of understanding him, as to the concepts and causative relationships, which he is not convinced existed, and which often result in a determination of irresponsibility and a life commitment, when a determination of responsibility but in need of special rehabilitative treatment for a relatively short time, would have been the realistic and better answer.

As to the accused who is found not guilty by reason of insanity under our proposal, his case should be required by law to be reexamined periodically in the following manner and through a consideration of his mental condition related to his potential danger to the peace and safety of the community, instead of a consideration of dangerousness alone as is now the case; under the present system, the acquitted and committed respondent's release, as stated above, is conditioned upon a statement signed by the Hospital Superintendent of "satisfactory proof that his discharge, or release, in the custody of a relative or friend, will not endanger the peace and safety of the public." This, then, requires that the Superintendent is clinically concerned and involved with individual patients, which is rarely the case, as can be well understood when one considers the size of our State Hospitals.

To eliminate the possibility of unnecessary and prolonged incarceration, we would like to see an *amendment to the law* to provide that a respondent, confined to a mental hospital by court order, must be reexamined once every year by a court-appointed psychiatric review board and that the board's findings must be referred to the court for its determination whether further confinement is necessary or desirable. This would eliminate the need for the Superintendent, a single individual, with vast administrative obligations and involvements, to take a stand in a matter that should be left to clinical judgment. In addition, it would relieve a Superintendent of the real and anticipated responsibilities and consequences of having to make predictions about the conduct of an individual in months and years to come – a divine task not available to psychiatrists.

A further objection to the statute relating to release or discharge of the committed respondent is to the requirement of the filing of a bond in the case of release to the custody of a relative or friend, the same provision existed in 1871 and at that time, perhaps, with some justification.

We find in many of the *few* cases in which release is authorized by the Court, subject to bond, that *the bond is not forthcoming,* due to the financial inability of the relative or friend to produce the bond, either personal surety or corporate; thus, we find that a provision apparently intended to facilitate release is, instead, a hindrance to it.

Certainly, the Court's decision to release is in no way based upon the protection availed the public by the bond, but is based upon the respondent's preparedness to return to the community. One hundred years ago, the bond provided some degree of reimbursement to the individual whose property or person was injured by the respondent after release; today, however, with the nearly universal coverage of insurance, e.g., health, accident, disability, life and casualty, this reason for the bond is of little significance. Further, and more importantly, there is little therapeutic value in saying to the releasee: "You are ready to resume your place in the community but someone else will be responsible for your acts."

We recommend release without bond.

In early 1965, as Maine moved toward completion of its planning in Mental Health, one of the groups assisting in formulating the plan, the Legislation and Forensic Psychiatry Task Force, comprised of attorneys, a psychiatrist, and a psychologist, devoted some time to the discussion of means by which the *Durham* dilemma might be resolved.

Schumacher[18] and members of the task force have recommended that the criminal procedure that leads to the establishment of guilt (or innocence) should precede a hearing on the absence or presence of illness, which would cause the respondent to commit the crime of which he has been convicted. In this manner, the presence of guilt would be proven first and replace the current plea of "not guilty" but if guilty, "I am not guilty because I am mentally ill."

If a respondent has been found guilty of a crime by due process of law and thereafter pleads that he is mentally ill, it has been previously proposed by one of the authors[19] that the Court should then refer this problem to a court-appointed review board of 3 qualified psychiatrists who should have the right and the resources to utilize the services of a qualified psychiatric social worker and a qualified clinical psychologist to assist in fact-finding and testing. The board should further be allowed to engage other specialists such as a clinical neurologist.

The diagnosis of psychiatric disorders is sometimes extremely complex and difficult and may require collaboration of several specialists. In this way, we could ensure that every respondent has a comprehensive and sound evaluation regardless of what his financial resources are and regardless of whether his defense counsel has superior skill and ability or not, whether he has been appointed by the Court.

This procedure, in our judgment, would constitute a vast improvement over present practices.

It would be incorrect, however, to anticipate that this would solve all problems.

We now submit that a better and more realistic solution is not in the dual hearing, particularly not if the

"Durham Rule" is retained, but would reiterate in conclusion:

1. The irresponsible will be exculpated by a return to M'Naghten.
2. The person who is aware of the criminality of his act who is otherwise mentally ill or mentally retarded can best be administered to in his interest, and in the interest of the public, by adhering to the theory "you are responsible for the commission of a criminal act but you need special rehabilitative treatment" which theory could be best implemented through the modernization of the system in Maine, i.e., construction of the proposed reception and treatment center and use of the center in part as a diagnostic facility, the latter to be used as an adjunct to the Court.
3. The sentencing laws should be modified along the lines of the Model Sentencing Act or similar provisions permitting the most beneficial use of psychiatric testimony in the diagnosis of the problems of the convicted respondent.

The psychiatrist would prescribe to the Court, at the time of sentence, as to the disposition of the case needed for the rehabilitation and treatment of the respondent, and if pertinent, for the protection of the public.

We recognize that our discussion is not unique, except as it may relate to Maine. We further recognize that few, if any, truly new furrows have been left on the ground we have covered; for, *Durham* has absorbed the attention of numerous writers, not to mention that of the Court of Appeals of the District of Columbia, all of which has resulted in volumes.

We do not submit this effort for the purpose of generating further prolonged discussion but for the purpose of prescribing a focal point for action oriented planning in Maine, *and elsewhere,* toward the end that the interests, purposes and efforts of the psychiatrist and allied professionals, the lawyer, the jurist, and the penologist, may be joined in the conduct of a truly functional criminal process acknowledging the needs of the convicted and acquitted respondent, and those of the people.

N.B. The views herein expressed are not necessarily the official views of Maine's Department of Mental Health and Corrections or Office of the Attorney General.

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Head and Neck Pain*

RUDOLPH W. PEARSON, M.D.**

To properly cover this subject, described in these four little words requires discussion by oral surgeons, otolaryngologists, orthopedists, neurologists, neurosurgeons and internists. But since we are a small group, each sharing with the other the every day problems of complaints from people who come in with pain in the head, we can settle down to reviewing a lot of facts that most of us know, some of the facts that all of us know, and I hope I might find one that might jog your memory or help in the settling of a problem in the future because we might have discussed it today.

I think it important to separate pain from headache because while all headaches are painful, not all pains are headaches.

Allow me to give an outline to help us think of these things in simple terms, though the subject certainly is complicated.

I) Pathways
- A) Nerves
  - 5 - 7 - 9 - 10
- B) Vessels
  - Carotid (1) external
    - (2) internal
    - (3) vertebral

II) Types
- A) Short
- B) Stabbing
- C) Sustained
- D) Prolonged
- E) Repeated
- F) Sustained

III) Functional
- A) Migraine
- B) Histaminic cephalgia (Horton's Syndrome)
- C) Tension

IV) Organic
- A) Tumors
  - 1) benign
  - 2) malignant
- B) Neuralgias
  - 5th, 7th, 9th, and 10th
- C) Tic - 5th and 7th
- D) Arthritis
  - 1) cervical spine
  - 2) cricoarytenoid joint

We shall talk about migraine and especially histamine cephalgia later. I think we can pass over the tumors of the brain and the meninges as causing headaches but not real pain usually. We can omit any ocular disturbance because it is stated by two very competent ophthalmologists that pain does not begin in the eye. Let us, therefore, begin at the bottom and work up, starting with the nerves most commonly involved. Certainly the tics, of which all of you are so familiar, are of the short, stabbing variety which are intense, debilitating, and sometimes described as an invitation to suicide. The methods of caring for these, of course, range from injection of the nerve or their ganglions with Novocain or Xylocaine® or Monocaine®, to alcohol injections, to actual division of the nerve which is sometimes a great relief in spite of the residual anesthesia. To section the ophthalmic branch of the fifth nerve, of course, is dangerous since it invites ulceration of the cornea, and sometimes loss of the eye. But one is occasionally driven even to this to get rid of the pain.

With the various facial tics, it is useful to remember that Dilantin®, 100 mg. t.i.d. is sometimes helpful in reducing the trigger mechanism which sets off the individual attacks. A new drug not yet fully reported, known as Tegratol®, in 200 mg. dosage is put out by the Geigy Chemical Co. Thus far it has not received approval of the FDA; and therefore, it is limited to usage not to exceed three months at any one time. We shall probably hear more of this chemical in the future since it is reputed to be almost specific for the tics.

Glossopharyngeal neuralgia is often overlooked and frequently confused with the fifth-nerve neuralgias but almost invariably begins in the region of the tonsil and radiates to the ear and the side of the head. This not infrequently can be found in association with pathology in and about the tonsil and/or the fossa. Don't overlook the possibility of malignancy within the tonsil, picked up not infrequently only by palpation and even more infrequently by a biopsy for complete investigation. This too can occur in the nasopharynx which must be meticulously examined and sometimes subjected to a blind biopsy. Beyond this, however, the styloid process and calcification of the stylohyoid ligament must be considered. Palpation again of the tonsil or its fossa will sometimes disclose an elongated styloid process or calcified ligament. If this appears to be the source of the pain and discomfort, sectioning of the muscles of the tonsillar fossa can be done, exposure of the styloid process made, muscle stripped from it, and a section of 1 cm. or more may be taken from the bone by the usual bone-cutting instruments. This is particularly so if the patient has had a tonsillectomy and complains of pain on swallowing starting at the time of a tonsillectomy and continuing with each meal since that date.

Pain in and about the cricoarytenoid joint must be noted. The patient's history suggests it by frequent attacks of hoarseness, unexplained on the basis of polyp or other pathology and of pain on swallowing or talking. Tenderness over the cricoarytenoid joint is very revealing, and limitation of the arytenoid on examination by mirror makes the diagnosis. The treatment, aside from

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that used to treat the arthritic processes in the remainder of the body, is augmented by manipulation of the joint and/or arytenoidectomy as described by Montgomery in his original work on this disease process. Glossopharyngeal pain usually implies radiation of pain into the ear, but beginning in the region of the tonsil or nasopharynx.

Hypopharyngeal pain, on the other hand, tends to be local but not infrequently causes enough pain in the ear to be a very distressing symptom. This is mostly found in neoplasms, usually carcinoma of the larynx which has become an extrinsic lesion involving the arytenoids and pyriform sinuses. This pain frequently radiates to the ear and can be controlled at times, once the diagnosis is made, by the application of cryotherapy. This is done with a laryngoscope, with or without general anesthesia and with the application of cold from 60-190° F. below zero, depending on the modality used. This is a very useful procedure in reducing the need for opiates from sometimes 300 or 400 mg. of Demerol®, for example, down to 50-100 mg. A day. Occasionally when the pain radiates from the hypopharynx to the ear, we have found it useful to anesthetize the great auricular nerve, as it comes out of the junction of the upper and middle thirds of the sternomastoid muscle; the result is very effective in reducing the otalgia. If this is successful and lasts only for a short time, then injection of alcohol will sometimes reduce the pain for a week or ten days, or more; and if this then fails, neurectomy through a simple incision over the posterior border of the sternomastoid and picking up the nerve can be very helpful. While this has little or no anatomical authority to recommend it, it is nevertheless an empirical means of reducing pain. It is rationalized by the neurologists as spilling over or a sparkgap of sensory energy from one nerve to the other.

In discussing arthritis or synovitis, let us not forget the temporomandibular joint with radiation of pain to the ear and to the temporal area, and along the face and jaw. It is sometimes of obscure etiology until one palpates the temporomandibular joint, tests the mobility of the jaw, examines the bite, and finds a defect in the closure of the jaw and in the structure of the teeth. If correction by exercises and adjusting the bite fail to help, injection of the joint with an intra-articular cortisone preparation, such as Kenalog® or Medrol®, can be extremely helpful. In fact, it is sometimes helpful to do this while the search for correction is being undertaken by the dentist or oral surgeon. I have illustrated the case of a fifty-two-year-old woman who, on the other hand, was thought to have a temporomandibular joint pathology but who later was found to have carcinoma spilling out from the medial wall of her antrum into her nose, eroding the four walls of the antrum and the gingiva and creating all of the pain. By the time that this was discovered, the patient's outlook was grave. A maxillectomy, but without exenteration of the orbit, was done. Biopsy on the operating table failed to show any evidence of tumor in the roof of the antrum. Surgery brought about only a temporary palliation. Later cobalt, and then finally chemotherapy were given in a vain attempt to save this woman's life.

Nasopharyngeal tumors, of course, should always be thought of. The benign ones such as the angiofibromata of the juvenile are not painful but are life threatening because of their predilection for bleeding. Persistent bleeding, however, in a middle-aged individual from the sphenopalatine area must always be carefully observed after the bleeding has been controlled, for the presence of tumor. So often the lymphoepithelioma or the undifferentiated carcinoma of the nasopharynx makes its first manifestation with nodes in the neck; but there are times when pain is created within the head and extension of the disease through the foramen lacerum or the foramen ovale may be the first signal of disease.

Let us remember then that the prolonged, sustained types of pain may be due to bone erosion, secondary to the primary lesion within the nose or throat, or even elsewhere. This must be respected and treated properly.

Now let us consider for a few moments the vascular types of headaches. These are divided basically into the migraine — the most common, certainly — and/or the histaminic cephalgia.

Migraine can be differentiated rather quickly and simply from histamine cephalgia or Horton's Syndrome by virtue of the fact that with migraine, the patient describes his inability to drive, of wanting to go home and bury his head in a pillow, and pulling down the curtain. The histamine cephalgia victim, on the contrary, jumps out of bed, paces the floor, sometimes pounds his head against the wall; and the woman will not infrequently get up in the middle of the night and iron or clean, or do some household chores which requires energy. Furthermore, the migraine almost never attacks during the night and never wakes anyone up; but histamine cephalgia will not infrequently wake people out of a sound sleep, send them bouncing out of their beds to seek relief in activity. Both are unilateral; but with migraine, nausea and vomiting, and sleep are the clues to the diagnosis; and with histaminic cephalgia, nausea seldom occurs, sleep never comes until there is relief, or until opiates have been given. The patient with migraine tends to look pale. The patient with histaminic cephalgia tends to have plethora on the side involved, with congestion and tearing of the eye, and congestion and obstruction of the nose on that side.

The treatment of migraine I'll leave to you. Everyone has his pet way of handling these things. Certainly they occur not infrequently as holiday headaches at the end of a period of tension, whether it be the end of the week, the end of an emotional strain, or the end of a long preparation for some exciting time. Doctor Alvarez of the Mayo Clinic tells us of the charming friend in Chicago who invariably insisted that he come have dinner with her in her apartment whenever he passed through Chicago. She was a meticulous person who laid a beautiful table with lovely silverware, flowers, and a tastefully prepared dinner. Unfortunately each time that

he got there, she was so glad to see him that she was in bed with a headache as a result of her preparations. This is typical of migraine.

On the other hand, histaminic cephalgia can occur with a peculiar individual timing. One patient of mine had episodes for six weeks each year, starting from the first of March until the middle of April, occurring each morning at ten minutes of three, no matter what time he went to bed, and no matter what he had done during the day. He was a very vigorous-living person, but was completely shattered with these headaches until I taught him how to give himself Gynergen® intramuscularly whenever they occurred. Within ten minutes he had relief. These are best treated, when they are prolonged, by hospitalization for one week for the intravenous use of histamine diphosphate. This is given by introducing one c.c. of 2.75 mg. of histamine diphosphate into 250 cc's of normal saline or dextrose and water. This is given daily for seven days, starting at 20 drops a minute for the first day and increasing by increments of 10 drops per day, up to 60 drops per minute for the fifth, sixth, and seventh days. During this time the patient becomes somewhat flushed, tends to have a constricting type of headache; and if this becomes too severe, of course, the treatment can be stopped or slowed down, and given at a lesser rate. Treatment in the hospital is followed by injections subcutaneously or intramuscularly twice a week for about four weeks, once a week for four weeks, and then once a month for several months. If the headaches tend to recur, the intramuscular treatments are stepped up to daily dosages for a week or two, and then gradually tapered off again.

In migraine, the use of Compazine® suppositories during the periods of nausea and vomiting is extremely helpful, using 25 mg. in the average adult; and this very often will abort the attack. During the day a person might use two cups of black coffee to abort the attack, or during the evening two shots of straight whiskey. A quick stimulant is very helpful. In the histamine cephalgia, however, such things as Bellergal® given daily if the attacks are frequent, or at twelve-hour intervals sometimes will abort or reduce the attacks in frequency or severity; and the use of Cafegot® at the very beginning of the attack sometimes is helpful in this direction. Sansert® is sometimes used in either one of these headaches on a daily schedule but must be used with considerable caution because of its alleged propensity to create retroperitoneal fibrosis. All of these drugs, of course, must be used with considerable caution.

Distribution of pain along the carotid branches, of course, is a well-known concept of vascular headache; and we musn't forget that these can be caused by allergies, especially food allergies. You note I haven't mentioned sinus pain, since this is a subject too familiar to dwell on: but the pathways of pain may be somewhat bizarre. For example, the patient with pain in the posterior ethmoids with pus coming from that area may have pain radiating from the region of the infra-orbital artery and nerve, up along the side of the nose, to the base of the frontal sinus. X-rays may prove all of these to be negative; but with careful inspection, aspiration and application of cocaine, etc. to the area involved, the diagnosis can be made. Furthermore, with vascular pain, Dr. Harris, an oral surgeon at Boston University, has proved to his satisfaction that some of the pain is carried along the capillaries and the gingiva in regions which are almost devoid of nerve supply. He has eliminated pain by pressure on the carotid artery, shutting off blood supply to the mandible and a consequent disappearance of pain. This was brought about by his investigation into the fact that sometimes novocain as a field block in the presence of infection fails to relieve the pain of which the patient complains. And we mustn't forget that the vertebral artery may carry pain. You are perhaps all aware of the vertebral-artery-occlusion syndrome which is alleged to create symptoms of labyrinthosis, suggesting Meniere's Disease. On turning the head to one side, occlusion of a vertebral artery which has a kink beneath the scalene muscles may provoke suppression of activity in the labyrinth on that side and a subsequent vertigo. On raising the hands over the head, a change in pulse may occur as a result of this same phenomenon or reflex consequent to it, which helps in the diagnosis. But we are interested primarily in pain which may radiate up along one occipital area and may be aggravated by turning the head to the left or to the right. An arteriogram is necessary to make the diagnosis; but the treatment is relatively simple, if one is familiar with neck surgery. Exploring the vertebral artery beneath the scalene and a myotomy of the scalene, releasing the kinked artery from its prison bed, brings welcome relief.

That brings us to the functional headaches; and these, of course, are relegated to the psychiatrist if they become severe enough. If you don't have a psychiatrist in your area, however, it sometimes is pertinent that you be the psychiatrist and at least give the opportunity to the patient to unroof some of the complexes and hidden problems of which I profess to know nothing. We can, however, encourage the patient to discuss it and relieve their minds of their depressions. A psychiatrist tells me that the hysterical types of pain are considered as a psychic solution to a problem; and it is here that the chase becomes exciting to one knowledgeable in this field.

Head pain may be tolerated but never enjoyed. The search will go on always in each individual because there are too many causes and too few cures.

Adrenal Crisis Precipitated by Dexamethasone Therapy in a Hypertensive Addisonian†

DAVID W. JAECKS, M.D.* AND GEORGE P. LORD, M.D.**

The occurrence of adrenal insufficiency during corticosteroid therapy is an arresting manifestation of iatrogenic disease.[1,2,3] The case reported here is that of a patient with limited adrenal reserve in whom severe arterial hypertension developed following institution of corticosteroid replacement therapy. Over a 12-year period the salt-retaining activity of his replacement regimen was progressively reduced in an effort to control the hypertension.[4] When, as a final step, dexamethasone (9 alpha fluoro-16 alpha methyl prednisolone-with a virtual absence of mineralocorticoid effect) was substituted for prednisone, the patient developed acute adrenal insufficiency which rapidly responded to DOCA (desoxycorticosterone acetate).[5]

Case Report

A 58-year-old Negro male entered the hospital in November, 1964 on his 50th admission, because of a syncopal episode.

In 1944 a left apical pulmonary infiltrate was first detected on chest roentgenograph. It was judged to represent inactive tuberculosis after cultures of sputum and gastric aspirate cultures were negative, and no change was detected in subsequent films. Blood pressure at that time was 140/105.

In the ensuing 6 years the patient was hospitalized 8 times with acute episodes of fever, chills, productive cough, vomiting, diarrhea, abdominal pain, and prostration. During one such admission, in 1950, abdominal pain and vomiting led to laparotomy; chronic cholecystitis and acute pancreatitis were diagnosed and the gallbladder was removed. Following this surgery sputum obtained at bronchoscopy was positive for Mycobacterium tuberculosis. Apical lordotic chest films and tomography revealed a left upper lobe tuberculoma which was treated with lobectomy, thoracoplasty, and a course of PAS (para amino salicylic acid) and dihydrostreptomycin.

At the time adrenal cortical function was investigated because of weakness, recurrent episodes of abdominal pain and vomiting, asthenic habitus, and low blood pressure (105/80). Pertinent studies are shown in Table 1. BMR was -16% and abdominal films showed no adrenal calcifications. The patient was given a daily supplement of 5 grams Sodium Chloride and transferred to a sanatorium.

In 1952 20 i.u. of ACTH was administered by intravenous infusion over an 8-hour period on two consecutive days. Urinary 17 ketosteroids were measured as 2.2 mg/24 hours control and 3.4 mg/24 hours on the second day. Shortly after this study the patient was readmitted semicomatose, hypotensive (60/30), febrile (39.1°C), and dehydrated with abdominal pain, distension, and vomiting. Diagnosis of Addisonian crisis was made, and dramatic improvement followed DOCA and cortisone therapy. After maintenance regimen of cortisone 25 mg. daily and two subcutaneous 125 mg. DOCA pellets was begun, his blood pressure was 140/90.

In 1954 the patient's blood pressure was noted to be significantly elevated for the first time, but returned to normal levels after several days of bed rest; intravenous pyelogram, electrocardiogram, and phentolamine test were normal. Corticosteroid replacement consisted of cortisone 20 mg. daily in divided doses, and DOC-TMA (desoxycorticosterone-trimethyl acetate) 12.5 mg. monthly.

By 1959, with blood pressure 180/120, early hypertensive retinopathy had developed, and the electrocardiogram showed left ventricular hypertrophy. Cortisone was reduced to 10 mg. daily, with 9 alpha fluoro hydrocortisone, 0.1 mg. on alternate days.

In 1961 frequent and severe bifrontal headaches and continued hypertension led to admission for reevaluation of therapy. A 5-day ACTH stimulation study performed while the patient was receiving dexamethasone 0.75 mg. and NaCl 4.0 gm. daily showed significant but reduced adrenal response (Fig. 1). A sodium deprivation test showed adequate ability to retain sodium on cortisone 20 mg. daily and the patient was discharged on this regimen (Fig. 2).

Throughout the next two years severe headaches continued and blood pressure averaged 210/130. Addition of Guanethidine 50 mg. daily and changing the replacement regimen to prednisone 5 mg. daily produced fair control of hypertension. In early 1964, hydralazine 200 mg. daily was added. Three weeks prior to admission the dosage of Guanethidine was increased to 60 mg. daily and one week later the patient began to experience orthostatic dizziness culminating in the syncopal episode on the day of admission.

Hospital Course

On admission, the patient was an alert Negro man chronically ill, complaining of dizziness, frontal headaches, and shortness of breath.

Blood pressure 180/130 sitting, 160/130 standing, pulse 110, temperature 36.8°C, respirations 30/min. Hypertensive and arteriosclerotic fundoscopic changes were present with generalized arterial narrowing, 4:1 venous-arterial ratio, a flame hemorrhage, marked venous nicking, and early copper wiring. The neck veins were not distended at 30°, but increased filling was elicited by pressure over the liver. Trachea was deviated 1 cm. to the left at the suprasternal notch. Tachypnea with an active, prolonged expiratory phase, dullness over the thoracoplasty, and bilateral wheezes and coarse rales were noted. Cardiac examination showed a thrusting PMI in the 4th intercostal space at the anterior axillary line, a loud S-4 gallop along the left sternal border, and a split, fixed P-2. A non-tender liver edge was palpable 6 cm. below the right costal margin; the spleen was not palpable; mild periumbilical tenderness was present. Pubic hair was normal in amount and distribution, prostate was normal in size, and the left testicle was slightly atrophic. Neither ascites nor peripheral edema were present. Neurological examination was within normal limits.

Admission Lab Data included hematocrit of 44%, leukocyte count 6,500 with 72% polymorphonuclear, 20% lymphocytes, 7% monocytes, and 1% eosinophils. Urinalysis: specific gravity 1.009, pH 5.5, protein 4+. Microscopic exam showed numerous WBC's.

Blood urea nitrogen was 27 mg.%, creatinine 1.6 mg.%,

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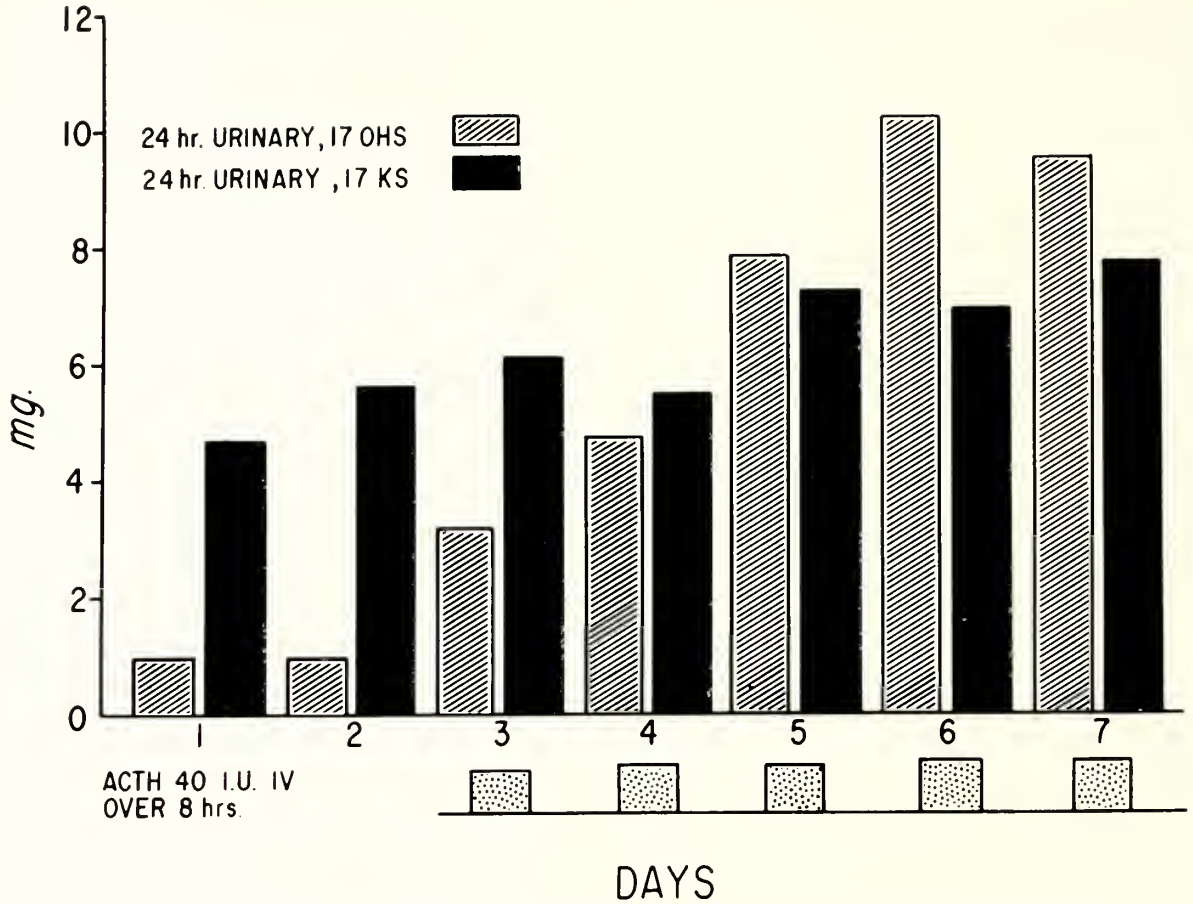


Fig. 1. Patient J. C. The response of urinary 17 keto- and 17 hydroxycorticosteroids during 5 days of ACTH stimulation. The baseline 17 KS and 17 OHS are low. During ACTH stimulation, 17 KS and 17 OHS increase but the response is subnormal.

FBS 97 mg.%, Na 138 mEq, K 4.4 mEq, Cl 97 mEq, CO_2 34 mEq.

A urine culture grew Klebsiella-aerobacter > 100,000 colonies/mm^3; sputum culture- E. coli.

Electrocardiogram showed left ventricular hypertrophy and lateral ischemia. Chest film: congestion and a small amount of fluid at the right base. Sinus films were normal and an IVP showed small kidneys bilaterally.

The initial regimen consisted of an 800 mg. (35 mEq) Na diet, SSKI, cyclopentamine and aludrine compound (Aerolone®) by nebulizer, guanethidine 40 mg., hydralazine 200 mg., prednisone 7.5 mg. and tetracycline 2.0 grams daily in divided doses.

During the first three hospital days his dizziness subsided and his dyspnea improved, but severe headaches continued together with nausea, vomiting, and loose stools twice daily. Blood pressure was 195/115. The headaches were ascribed to hypertension, and since mineralocorticoids potentiate development of hypertension, it was decided to begin a steroid regimen devoid of mineralocorticoid activity. On the fourth hospital day prednisone was stopped and dexamethasone 1.0 mg. daily begun (Fig. 2).

By the seventh hospital day blood pressure had stabilized at 140/80, hydralazine was discontinued, but the patient was still symptomatic, having headaches, loose stools, and occasional vomiting, but he was alert, ambulant, eating well, and maintaining his body weight. On the eleventh hospital day he vomited several times and complained of increased fatigue and epigastric pain. Later he was found lethargic, delirious, and in moderate respiratory distress. Pulse 48, BP 160/90, venous pressure 230 mm. saline in the antecubital vein. Electrocardiogram showed absence of atrial activity, nodal rhythm, and tall slender inverted T waves.

Serum potassium was 7.1 mEq, sodium 127 mEq, carbon dioxide combining power 29 mEq, and chloride 90 mEq (Fig. 3), 300 cc. of 50% glucose with 35 units regular insulin was started by i.v. infusion immediately, followed by Kayexalate® (cation exchange resin) 50 gms. with D-sorbitol 200 gms. by enema. The serum potassium fell to 6.7 mEq over the next 12 hours but the patient worsened, becoming comatose with a divergent squint, at which time the blood pressure was 130/80 and the blood glucose 86 mgms%. Hydrocortisone 50 mg. was given i.v. and Kayexalate 100 gms. by enema with a resultant reduction of serum potassium to 6.1 after another 12-hour period. Some concomitant clinical improvement occurred, but the patient remained obtunded and very weak. DOCA 5 mg. i.m. was then administered, and within 14 hours the serum electrolytes were within normal limits (Na 141, K 4.1, CO_2 35, Cl 92), and the patient was alert and ambulatory. Over the following week the electrocardiographic changes resolved, and no elevation of white blood count, sedimentation rate or SGOT developed.

He was again placed on prednisone 7.5 mg. daily and was discharged on this dose with a BP in the 180/100 range.

Discussion

The diagnosis of adrenal insufficiency in this patient

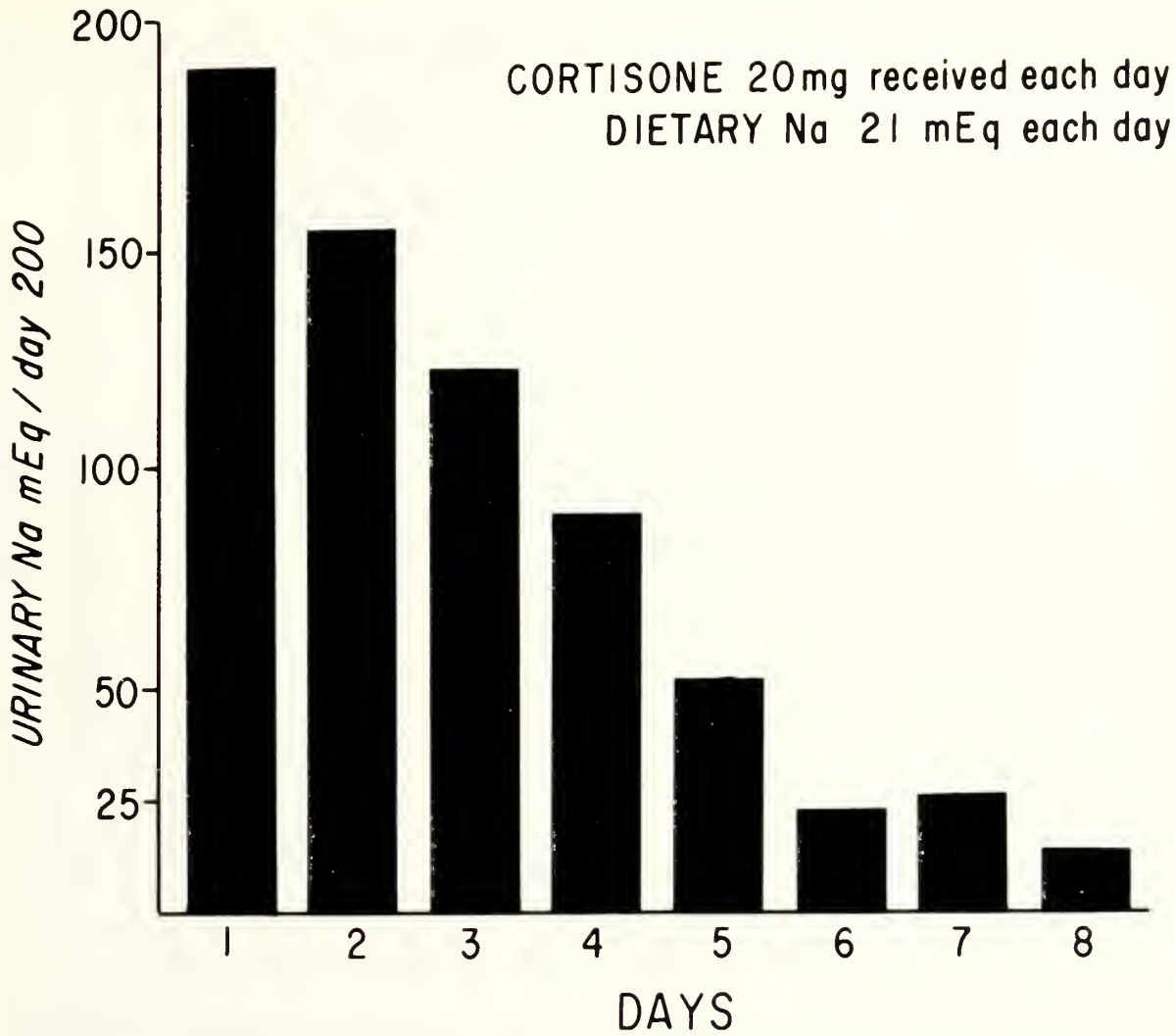


Fig. 2. Patient J. C. Maintenance of cortisone but restriction of sodium intake demonstrates this patient's ability to decrease sodium excretion in the face of sodium restriction. However, the net sodium balance was negative until day 6 as a manifestation of limited sodium retaining ability.

is well supported by the clinical and laboratory data. The initial adrenal function studies (Table 1) are indicative of reduced but not absent adrenal function. Especially noteworthy in this regard is the borderline low *24 hr. 17 KS* determination in 1950 and the small rise measured after two days ACTH stimulation.[11] The presence of slight endogenous adreno-cortical function was further indicated by small delayed rise in urinary 17 hydroxy and 17 keto steroids with 5 days of ACTH stimulation (Fig. 1).

In addition to the laboratory data, the clinical presentation with vomiting, fever, prostration, and hypotension preceding institution of steroid therapy is strongly suggestive of adrenal crisis. That adrenal insufficiency in this patient is not secondary to pituitary insufficiency is suggested by the absence of signs of hypogonadism or hypothyroidism. There are no visual field defects or changes in the sella turcica suggestive of pituitary tumor. An FSH determination recently was low, but low FSH is found associated with chronic disease and this patient has had chronic pyelonephritis and chronic bronchitis, in addition to the diseases mentioned above.

The etiology of this patient's adrenal insufficiency is most likely due to adrenal tuberculosis or to partial adrenal atrophy. There is a temporal coincidence between the discovery of active tuberculosis and adrenal insufficiency in this patient, but tuberculosis was present at least 6 years before the adrenal insufficiency became manifest. Abdominal calcifications were not seen, but this is often true in Addison's disease of proven tuberculosis etiology.[3] The underlying adrenal insufficiency may have superimposed pituitary-adrenal incompetence, induced by 12 years of replacement therapy, although 3 years of replacement corticosteroids in normal adults was shown to have no lasting effect on pituitary function.[1]

Hypertension in Addison's disease is of two general types: the first, related to excessive DOCA or mineralocorticoid replacement therapy,[14] and second, a manifesta-

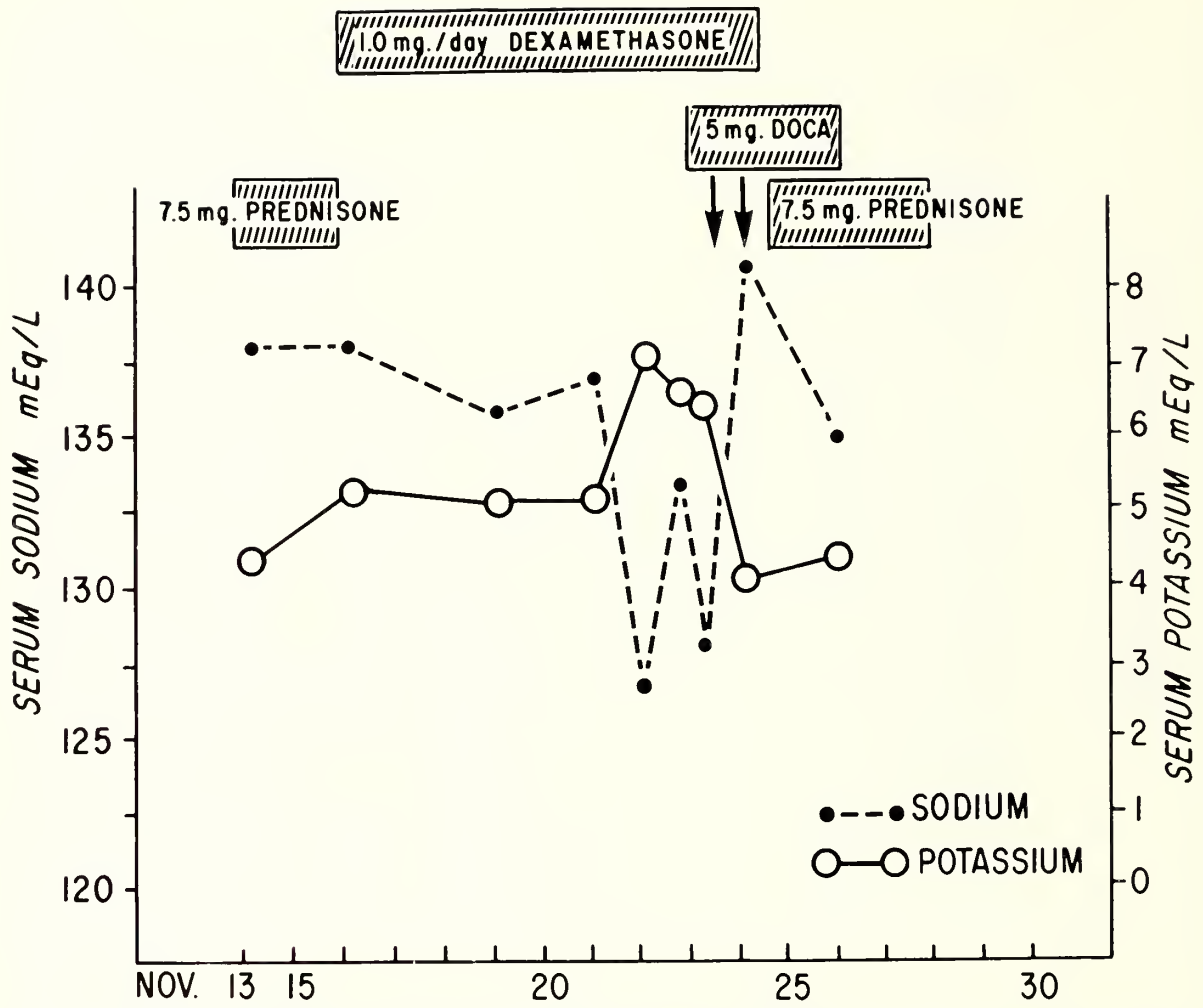


Fig. 3. In this patient with limited adrenal reserve and hypertension, the substitution of Dexamethasone for Prednisone, in an effort to control hypertension, resulted in a precipitous fall in serum sodium and a rise in serum potassium after 6 days.

tion of an endogenous hypertensive mechanism such as eclampsia[15] or renal cortical infarction.[16] In this patient the presence of persistent pyuria, proteinuria, positive urine cultures and small kidneys by IVP make chronic pyelonephritis a likely mechanism. Some patients with Addison's disease have high circulating renin levels.[17]

The question of steroid replacement therapy for the hypertensive Addisonian focuses upon the interaction of the adrenal cortex and the sympathetic nervous system.[18] Many of the actions of circulating epinephrine cannot be elicited in the absence of steroids. Addisonian patients with hypertension have vascular disease rendering them unusually sensitive to the pressor effects of DOCA on the one hand[19] and more sensitive to stress than patients with adrenalectomy for other causes, on the other.[20]

Compared to cortisone, dexamethasone has virtually no sodium retaining properties with the usual assay techniques.[21] Patients on adrenal replacement therapy with this medication may be Cushingoid in appearance while in adrenal crisis.[22] If secondary to chronic pyelonephritis, one explanation of the hypertension in this patient is a reduction in renal blood flow with stimulation of juxtaglomerular cells, increased renin production and activation of the renin-angiotensin system. In addition to its direct effect upon the vascular system, Angiotensin II in normal man stimulates the zona glomerulosa with elaboration of aldosterone. The renal tubules respond with increased sodium retention, expansion of circulating blood volume and an increase in renal blood flow.[23]

The following sequence of events is proposed to explain the development of adrenal crisis in this patient. Dexamethasone blocked pituitary ACTH production. Since aldosterone production in the adrenal is partly dependent on ACTH the ability to retain sodium by endogenous aldosterone production was inhibited. Since dexamethasone by itself did not cause sodium retention, the patient was thrown upon the intrinsic mechanism of his kidneys to do so. Years of hypertension, chronic infection and the concurrent administration of hypotensive

agents rendered his kidneys incapable of maintaining electrolyte homeostasis and crisis supervened.

This experience dramatically underscores the difference in synthetic adrenal cortical preparations and the sensitivity of the hypertensive Addisonian to a change in steroid environment.

Summary

This is a case report of a severely hypertensive patient with Addison's disease. Steroid replacement therapy was altered from cortisone to prednisone and finally to dexamethasone in an attempt to ameliorate the hypertension by decreasing salt-retention. While blood pressure did fall, after 11 days of dexamethasone therapy, adrenal crisis supervened. This report emphasizes the danger of administering non salt-retaining steroids in the treatment of adrenal insufficiency.

Acknowledgment

The authors wish to thank Dr. Seymour Reichlin for his review of the manuscript.

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Cardiopulmonary Resuscitation at a Community Teaching Hospital — Report of 37 Cases†

GUNTER L. SPANKNEBEL, M.D.* and PURUSHOTTAM A. KALE, M.D.**

INTRODUCTION

Cardiopulmonary resuscitation by external cardiac compression and expired air ventilation has become accepted by the profession in the treatment of unanticipated "sudden death." The basic principles of cardiopulmonary resuscitation developed and tried at large medical centers are being translated into terms applicable to community hospitals.

This report describes the results of the cardiopulmonary resuscitation program started in July 1964 at the Memorial Hospital, Worcester, Massachusetts. Certain criteria are proposed for evaluation of results of the resuscitation. The report also emphasizes the impact of training and individual experience on the success of such a program.

DEVELOPMENT OF THE RESUSCITATION PROGRAM

Prior to July 1964, only two cases of successfully resuscitated patients with cardiopulmonary arrest were described in the case records of the Memorial Hospital. A plan of action in the event of cardiac arrest did not exist.

In July 1964 a cardiac resuscitation committee, consisting of a cardiologist, a thoracic surgeon and an anaesthesiologist was appointed and upon the arrival of the new intern and resident staff an intensive training program was begun.

MATERIALS AND METHODS

The "plan of action in cardiac arrest" outlined by Johnson[1] was adopted with following modifications:

1. "Dr. Haste to . . . (location)" was announced over the paging system in case of an emergency. This way of paging alerted the hospital medical and paramedical staff only. The first trained person to arrive on the scene started at once with the proper resuscitation measures. Within one minute a complete, well-trained resuscitation team could usually be formed out of the immediately available house staff.

2. The chief medical and surgical residents and the cardiology resident were always available to take charge of treatment.

3. The resuscitation equipment was transported to any area in the hospital well within five minutes of the emergency page. It consisted of a D.C. defibrillator, cardiac monitor and pacemaker (Lown Cardioverter Table Model 10530B, American Optical Company), thoracotomy-, tracheostomy-, cutdown- and subclavian vein puncture sets, laryngoscopy and intubation equipment, airways, breathing bag and mask. A separate cart contained all necessary intravenous fluids and pharmacological agents, intravenous and intracardiac needles and tubing.

4. Subclavian vein puncture was performed as described by Davidson et al[2] unless an intravenous route had already been established prior to arrest.

5. Running notes were dictated by the doctor in charge of the attempt and a comprehensive chronological record was made later on the chart.

6. In a successful attempt the patient's state was monitored for at least 48 hours by the house staff.

The result of the treatment was classified as follows:

1. *Clinical Success:* Recovery of cardiopulmonary and central nervous system functions for at least four hours.

2. *Technical Success:* Return of cardiopulmonary, but no return of central nervous system function with the patient remaining alive for at least four hours; or a complete clinical recovery for less than four hours.

3. *Failure:* Cases not included in the above two groups.

The effectiveness of the program was evaluated by analyzing the results of the first and the second half of the study period separately (Table 2).

RESULTS

Forty-three episodes of cardiopulmonary arrest in 37 patients were treated from July 1, 1964 to June 30, 1965 (Table 1).

There were 21 male and 16 female patients from 49 to 83 and from 55 to 81 years old, respectively.

In eight patients nine attempts were "clinically successful." Patient No. 19 suffered two cardiac arrests and was resuscitated successfully each time. Patient No. 28 had a second fatal cardiac arrest 37 hours after the first clinically successful resuscitation. Seven of the eight patients went home clinically well and are alive 3-8 months later at the time of writing this report.

Only three patients had the arrest in the operating room (No. 5, 31, 32); the remainder were treated in various wards of the hospital. This was encouraging considering the pre-arrest status of the patients. During

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the second half of the study period the number of technical successes decreased, the clinical success rate mounted, (Table 2) indicating a higher survival rate of potentially salvageable patients, with growing experience of the house staff and improvement in techniques of resuscitation.

TABLE 1

ATTEMPTS AT CARDIOPULMONARY RESUSCITATION

July 1, 1964–December 31, 1964

(Cases 1-17)

Case No.	*Sex*	*Age*	*Diagnosis*	*Cause of Arrest*	*Post-arrest stay*	*Deficiencies*	*Result*
1	F	67	severe diffuse coronary sclerosis; atrophy of thyroid*	ventricular fibrillation	0	intubation; pharmacologic management	failure
2	M	59	idiopathic cardiac enlargement; cardiac cirrhosis; hyperkalemia	cardiac standstill	1 hour	intubation; internal pacemaker	failure
3	M	74	severe aortic stenosis; coronary arteriosclerosis*	slow idioventricular rhythm	1 hour	internal pacemaker; pharmacologic management	failure
4	M	83	old posterior myocardial infarction; hypertensive cardiomegaly; pulmonary edema; bilaterial hydrothorax*	ventricular fibrillation	1 hour	intubation; internal pacemaker	failure
5	F	78	atherosclerotic aneurysm of abdom. aorta with rupture; hypertensive cardiomegaly*	asystole on operating table; ventricular fibrillation	2 hours postop.	monitor; pacemaker; pharmacologic management	failure
6	M	76	old myocardial infarction; atherosclerosis; strangulated left direct inguinal hernia; bronchopneumonia; pulmonary embolus + infarct; hydrothorax; acute ulcers of stomach*	ventricular fibrillation	9 hours	pharmacologic management	technical success
7	M	49	extensive myocardial infarction*	cardiac asystole	0	pharmacologic management internal pacemaker	failure
8	M	72	coronary insufficiency	ventricular fibrillation	4 hours 25 minutes		technical success
				second arrest ventricular fibrillation	0		failure
9	F	63	acute myocarditis*	ventricular tachycardia	1 hour		failure
10	M	81	severe coronary sclerosis	ventricular tachycardia; asystole	2 hours 40 minutes	internal pacemaker; pharmacologic management	failure
11	F	58	state post abdominoperineal resection for adenocarcinoma of rectum; transitional carcinoma of bladder neck; coronary arteriosclerosis*	cardiac standstill	1 hour 10 minutes		failure
12	F	70	aortic stenosis; pulmonary edema*	cardiac asystole	1 hour	pharmacologic management; internal pacemaker	failure

Case No.	*Sex*	*Age*	*Diagnosis*	*Cause of Arrest*	*Post-arrest stay*	*Deficiencies*	*Result*
13	F	68	perforated diverticulitis; myocardial infarction; congestive heart failure	ventricular fibrillation	5 hours	internal pacemaker	technical success
				cardiac asystole	0		failure
14	F	79	myocardial infarction; inner third of left ventricle*	ventricular fibrillation	0	pharmacologic management; internal pacemaker	failure
15	M	79	myocardial infarction	"cardiac arrest" (clinical diagnosis)	2 mo.		clinical success
16	M	69	myocardial infarction	Stokes Adams; cardiac asystole	9 hours 10 minutes	internal pacemaker	technical success
				second arrest asystole	0	internal pacemaker	failure
17	M	51	myocardial infarction	ventricular fibrillation	1 hour	delayed use of defibrillator; internal pacemaker; effective massage	failure
			January 1, 1965–June 30, 1965 (Cases 18-37)				
18	F	58	myocardial infarction	ventricular tachycardia; standstill	1 hour	internal pacemaker	failure
19	M	59	myocardial infarction	ventricular fibrillation	26 hours		clinical success
				ventricular fibrillation	2 mo.		clinical success
20	M	79	massive pulmonary embolism*	"cardiac arrest" (clinical diagnosis)	0	effective oxygenation	failure
21	M	65	abdominal aortic aneurysm; cystitis; acute myocardial infarction	ventricular fibrillation	0	internal pacemaker	failure
22	M	59	acute myocardial infarction*	ventricular fibrillation	0	? internal massage	failure
23	M	56	ASHD: acute myocardial infarction	complete heartblock changing to ventricular fibrillation	0		failure
24	M	51	acute myocardial infarction	ventricular fibrillation cardiac standstill	0		failure
25	F	74	multiple pulmonary emboli; hypertensive cardiomegaly; localized myocardial fibrosis*	cardiac standstill	4 hours		technical success
26	F	70	acute myocardial infarction; pulmonary edema; adenoma thyroid and adrenals*	cardiac standstill ventricular fibrillation	0	defibrillation; pharmacological management	failure
27	M	68	extensive acute myocardial infarction	ventricular tachycardia	0		failure
28	M	53	myocardial infarction	ventricular fibrillation	37 hours		clinical success
				ventricular fibrillation	0		failure
29	M	64	calcific aortic stenosis; myocardial fibrosis*	asystole	0	effective CNS circulation; ? emergency valvotomy	failure

Continued on Page 275



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Case No.	*Sex*	*Age*	*Diagnosis*	*Cause of Arrest*	*Post-arrest stay*	*Deficiencies*	*Result*
30	F	81	acute myocardial infarction; renal insufficiency; bronchopneumonia*	ventricular fibrillation	0		failure
31	F	64	ASHD; angina pectoris; acute myocardial infarction; 2 yrs. postop. for sigmoid Ca; 2 yrs. post-nephrectomy	ventricular fibrillation	14 days		clinical success
32	M	82	embolus to right femoral artery	asystole; Stokes Adams	26 days		clinical success
33	M	68	cancer of prostate with metastases; mitral regurgitation; ASHD; urinary tract infection; anemia	ventricular fibrillation	32 days		clinical success
34	F	75	post-colostomy obstruction; Ca. of anus (1961); post-abdominal perineal resection	"cardiac arrest" (clinical diagnosis)	6 days	absolutely poor record of attempt	clinical success
35	F	75	hypothyroidism post thyroidectomy; hypoparathyroidism; ASHD; atrial fibrillation; angina pectoris	"cardiac arrest" (clinical diagnosis)	3 weeks		clinical success
36	F	80	acute myocardial infarction; prerenal azotemia due to prolonged cardiogenic shock	asystole	0		failure
37	F	55	severe coronary arteriosclerosis; congestive failure; repeated myocardial infarctions in the past.	ventricular fibrillation	1 hour	pharmacologic management; internal pacemaker	failure

*Autopsy performed

TABLE 2

EFFECT OF INDIVIDUAL EXPERIENCE ON SUCCESS RATE

(approximate %)

Period	*Number of Patients*	*Number of Arrests*	*Clinical Success*	*Technical Success*	*Failure*
July 1 to December 31, 1964	17	20	1 (5%)	4 (20%)	15 (75%)
January 1 to June 30, 1965	20	23	8 (35%)	1 (4%)	14 (61%)
TOTAL	37	43	9 (21%)	5 (11.6%)	29 (67.4%)

ILLUSTRATIVE CASE REPORTS

Case No. 19: This 59-year-old, white, male bank employee was admitted to the hospital on January 1, 1965 with a typical history of acute myocardial infarction. Physical examination was within normal limits, except for frequent extrasystoles. The EKG was consistent with acute anterior myocardial infarction. Other investigations, including enzymatic studies, corroborated this diagnosis.

The patient was treated with rest, anticoagulants and sedatives. On the 11th hospital day, at 2:55 P.M., he was found to be unconscious, cyanotic, without pulse, blood pressure or audible heart sounds. External cardiac massage and inflation bag breathing was started within one minute. Intravenous medications were administered through a right subclavian vein puncture. Intracardiac adrenaline and calcium chloride was given while the subclavian route was being established. The cardiac monitor was connected at 3:05 P.M. and showed ventricular fibrillation (VF). A single DC countershock of 200 w/second restored normal sinus rhythm (NSR) for a brief period, but VF recurred. Another shock of 300 w/second was given. The rhythm reverted back to NSR for a brief period, but went back to ventricular tachycardia (VT). Two hundred mg. of intravenous lidocaine hydrochloride (Xylocaine®) and a single DC countershock of 400 w/second were given. NSR returned with a blood pressure of 76/- and a pulse of 110 per minute. Patient was not cyanosed and started moving his eyelids at 3:12 P.M. A systolic blood pressure of 110 mm. Hg was maintained with norepinephrine (Levophed®) drip. At 3:45 P.M. VT occurred with a rate of 200 per minute. Two hundred mg. of lidocaine hydrochloride was given IV and a drip with 2,000 mg. lidocaine in 1,000 cc. of 5% dextrose in water was started without any effect on rhythm. Two successive countershocks of 400 w/second were given and NSR returned. Blood pressure was 140/70 mm. Hg. Patient was conscious but drowsy and responding to orders sluggishly. Urine output was checked every hour with an indwelling catheter. At 4:15 P.M. patient

was fully conscious. At 4:30 P.M. VT recurred. Two hundred mg. of lidocaine hydrochloride was given intravenously without effect, but a single DC countershock of 400 w/second reestablished NSR. Patient was conscious throughout. At 4:43 P.M. VT recurred. This time 0.1 g. of quinidine gluconate was given IV but there was no response for one minute. After two consecutive DC countershocks of 400 w/second the rhythm reverted to normal sinus mechanism with a blood pressure of 120/80. Patient was fully conscious. EKG showed frequent ventricular premature beats (VPB). Lidocaine drip was continued at a slow rate and the ectopic focus was successfully suppressed. Blood chemistry remained within normal limits. The pH was kept in normal range by frequent administration of sodium bicarbonate. At 8:00 P.M. tracheostomy was performed and a cuffed tracheostomy tube inserted. The patient was alert, moved all limbs, had a good urine output, bowel movement, and maintained his blood pressure without norepinephrine. He was taking general liquids by mouth and appeared to be over the crisis. The lidocaine drip was discontinued.

Twenty-six hours after the first arrest at 4:45 P.M. he had another episode of cardiac arrest with monitor showing ventricular fibrillation. Sequence of events was as follows:

4:55 P.M., 400 w/second single DC countershock – NSR – VPB – VF.

5:01 P.M., 400 w/second countershock – NSR.

5:06 P.M., VT.

5:07 P.M., NSR – good cardio-respiratory function, pupils reacting normally to light.

5:11 P.M., VT – cardioversion with synchronized countershock of 400 w/second – NSR with VPB – NSR. Blood pressure 70 mm. systolic. 5 mg. of metaraminol bitartrate was injected intramuscularly.

Patient unconscious. Respiration maintained with intermittent positive pressure breathing apparatus.

6:14 P.M., patient started moving the extremities. Decerebrate rigidity with involuntary spastic movements of arms and bilateral Babinsky response. Vital signs were stable.

10:00 P.M., patient breathing on his own.

Patient remained unconscious with bilateral Babinsky for 48 hours. Then slight progressive improvement was noted for the next 48 hours.

Five days after the second arrest there was no further improvement in his CNS status except for a positive menace reflex.

The patient was restless and semiconscious on the seventh post-arrest day. The tracheostomy tube was withdrawn. He was aphasic but conscious on the tenth and uttered a few words on the twelfth post-arrest day. The improvement in his general condition from this point on was more rapid and progressive. One month after the second cardiac arrest an electroencephalogram was consistent with mild diffuse cerebral damage. At time of discharge two weeks later the patient was ambulatory with unimpaired CNS functions, except for a slight memory defect for the events just prior to and at the time of arrest, and a slightly slurred speech. Presently the patient is completely ambulatory and alert and carrying on normal activities.

Comments: The case is noteworthy for the multiplicity of defibrillation attempts, two episodes of cardiac arrest and apparently remarkable recovery of overall central nervous system functions after the second cardiac arrest.

Case No. 31: This 64-year-old Turkish woman was admitted to the Memorial Hospital on March 12, 1965 for closure of a colostomy. A cancer of the sigmoid colon had been resected two years previously. She was a known case of atherosclerotic, hypertensive heart disease and gout and had suffered a myocardial infarction five months previously. Her past history was significant for thyroidectomy and nephrectomy. She was on treatment with digitalis, chlorothiazide and low salt diet.

Physical examination revealed a blood pressure of 200/100, apical pulse 95/min. irregular. Neck veins not engorged. Left carotid artery tortuous and dilated, but without bruits. Heart was enlarged with evidence of left ventricular hypertrophy, a grade 2/6 loud mid-systolic murmur at apex, a ringing second sound in the aortic area and a grade 2/6 early diastolic murmur in the third left interspace parasternally. There was a large incisional hernia in the left flank (post-nephrectomy). Trace of pitting edema was noted on both ankles.

A chest film revealed generalized cardiomegaly and bilateral pulmonary vascular congestion.

An electrocardiogram was consistent with left ventricular hypertrophy, atrial fibrillation, digitalis effect and anterior infarct.

Patient had frequent episodes of chest pain during her hospital stay. Ventricular fibrillation occurred during induction of anaesthesia on the 27th hospital day. Immediate external cardiac massage and ventilation through an endo-tracheal tube was started. A single DC countershock of 250 w/second reverted the rhythm to normal sinus mechanism. Cyanosis disappeared, normal reaction of pupils returned and the blood pressure rose to 210/110. At the onset of multiple ventricular premature beats, 200 mg. of lidocaine hydrochloride was injected intravenously, but an episode of ventricular tachycardia occurred seven minutes later. This was reverted to NSR by a synchronized countershock of 250 w/second. An intravenous infusion of 1,000 mg. of lidocaine in 500 cc. of 5% dextrose in water was given at a rate sufficient to suppress ventricular ectopic rhythm. Acidosis was combatted wih 44.6 mEq. of sodium bicarbonate intravenously every five minutes until effective spontaneous cardiopulmonary function was reestablished. Patient was conscious one hour later and had an uneventful hospital course. She was discharged on the 40th hospital day, ambulatory, on limited activity and with her clinical condition back to pre-arrest level. She showed no evidence of cerebral damage and is doing well at present.

Comments: The case is remarkable considering the past history. Emergency resuscitative treatment was given and continued because of the prompt availability of the means and the apparently complete removal of the tumor at the time of previous operation. Her response to the treatment was both prompt and dramatic.

Discussion

The prompt treatment of cardiopulmonary arrest needs no comment, but proper coordination of and guidance in all team efforts needs to be emphasized. The chief residents and the cardiology resident are best suited to fulfill the responsibility, since at least one of them is always immediately available.

In view of the limited time available for positive action, initiation of treatment was vigorous in each case of cardiopulmonary arrest. Continuation of the resuscitative measure, however, was then guided by the state of the patient's disease.

Case No. 33 was a patient with untreated carcinoma of the prostate with bony metastases, but resuscitative measures were continued because hormonal therapy was expected to give long-term alleviation. In Case No. 31 the apparently complete removal of a tumor in the past and the prompt availability of resuscitative help led us to continue the treatment to a successful recovery. The scope for selection of patients thus becomes wider and will change further as new and more effective methods of therapy become available for the presently "incurable" diseases. Hence the decision of offering the emer-

gency help cannot be based on generalizations or classified criteria.

The immediate delivery of drugs to heart and body tissues is a vital part of the management, so that establishment of a reliable intravenous route is imperative. Peripheral venipuncture with a sufficiently large needle is many times not possible. Venotomy requires valuable time and working space. Both routes are questionably effective, because external cardiac massage produces a poor blood flow at best despite a satisfactory pulse and blood pressure.[4] Intracardiac injection is effective, but cannot be performed repeatedly over a short period of time.

Percutaneous subclavian vein puncture and insertion of a plastic catheter, however, can be accomplished readily and with a low incidence of complications.[2,3] This has the advantage of rapidly establishing a reliable intravenous route through a deep vein proximal to the heart. Additional benefits are easy monitoring of the central venous pressure and reduced hazard of the catheter being dislodged during voluntary or involuntary movements of the limbs by the patient.

The need for some type of emergency internal cardiac pacemaker was felt in 14 cases. External cardiac pacemaker was of little help in initiating effective cardiac action in this series. Himmelhoch et al[5] also reported a uniform failure of external cardiac pacemaker, but Zoll and Linenthal[6] found this to be an effective way in many of their cases.

Use of lidocaine hydrochloride in suppression and prevention of ventricular arrhythmias was reported by Harrison et al[7] to be superior to procaine or procainamide as the latter causes more marked myocardial depression and more consistent fall of blood pressure. Quinidine is possibly a satisfactory drug, but ventricular arrhythmias and death in patients on quinidine therapy are reported.[8] The onset of action of lidocaine is prompt and the duration of its antiarrhythmic effect is brief; 10-20 minutes. Its action can be readily controlled by continuous intravenous drip. In doses described by Harrison et al,[7] it was a valuable therapeutic adjunct in this series.

Patient No. 28 had a brief convulsive seizure following administration of lidocaine intravenously. However, its relationship to the administration of the drug was not definitely established. The seizure was easily controlled by intravenous administration of sodium luminal and the treatment of arrhythmia with lidocaine could be continued without further seizures.

Pearson and Redding,[9] based on their experimental and clinical studies, advocated immediate administration of epinephrine to all patients with cardiac arrest, irrespective of the underlying disturbance of rhythm. This was also found useful in the present series.

The treatment of metabolic acidosis with sodium bicarbonate poses a problem in itself, especially in patients with myocardial infarction. Significant quantities of sodium administered intravenously at a rapid rate increase the cardiac load and diminish the chances of ultimate recovery. The favorable reports of the use of a new buffering agent THAM offer new hope in the treatment of this complication.[10]

Sustained ventilation through an endo-tracheal tube can be irritating to the patient, especially when he regains consciousness. Tracheostomy and introduction of a cuffed tracheostomy tube reduces the discomfort and prevents the regurgitation of pharyngeal and gastric secretions in the tracheobronchial tree.

A tabulated record of events and treatment at the time of each resuscitative attempt is always desirable but not often possible. Hence a system of taking down "running notes" dictated by the doctor in charge was found helpful in such an emergency.

Finally, it was of interest to note the improvement in the survival rate of cardiac arrest patients during the second half year of the resuscitation program (Table 2). This was felt to be due to proper training and increasing individual experience of the house staff.

Summary

1. Forty-three episodes of cardiac arrest occurring in 37 patients are reported.

2. Criteria for evaluating the results of resuscitation are suggested and results grouped under "clinical success," "technical success" and "failure."

3. There were nine "clinical successes" in eight patients (21%), five technical successes (11.6%) and 29 failures (67.4%). One patient was successfully resuscitated twice from cardiac arrests occurring at an interval of 26 hours. Seven of the eight patients were discharged from the hospital and are alive and well 3-8 months after their episodes of arrest.

4. With growing individual experience and improvement in resuscitative techniques, the survival rate improved from 5% to 35%.

5. Advantages of subclavian venipuncture are stressed.

6. Administration of lidocaine hydrochloride for control of ventricular arrhythmia was found to be effective.

7. The scope of patients to be offered emergency resuscitative measures is widening and the decision to continue them in a particular patient should be based on evaluation of multiple factors.

Acknowledgment

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Effect of Drugs on Heart Sound Intensity†

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INTRODUCTION

In an attempt to learn something of the hemodynamic factors which may alter heart sound intensity, several drugs were given to patients to alter circulatory hemodynamics. Because in the human subject it was not possible to isolate single hemodynamic factors in the presence of changes in other factors, no conclusion regarding the effect of single factors on the intensity of heart sounds can be drawn. However, the effects of the drugs on the heart sounds are clear-cut and of interest by themselves. Additionally, inferences regarding the alteration of hemodynamics associated with the heart sound changes may hold important clues for pinpointing future basic investigation in this area. In the meantime, until suitably controlled animal studies are performed, the physician may find some of the inferences from our study interesting and useful in assessing his patient.

Because of their rather diverse and opposing effects, the drugs chosen for study were: Amyl Nitrite, Methoxamine and Norepinephrine.

MATERIALS AND METHODS

Studies were performed on 20 middle-aged male patients, 15 of whom had normal cardiovascular systems. One had atrial fibrillation, the others, sinus rhythm. Using amyl nitrite, observations were made on 20 patients; 16 were studied with methoxamine and 8 with norepinephrine. Methyprylon, 300 mg., was given orally before each study. Retrograde catheterizations with percutaneous femoral artery puncture were performed using the Seldinger technique.[1] A #260 polyethylene loop end catheter[2] was placed in the ascending aorta or left ventricle. Pressures were obtained either by a Statham (23Db) transducer with a Midwestern photographic recorder at a paper speed of 150 mm/sec. or by a Schwarzer transducer and Schwarzer direct writing recorder at a paper speed of 100 mm/sec. Both systems produced equally satisfactory tracings. The natural resonance of the catheter-transducer system used was seven cycles per second and critical damping gave a flat response to thirteen cycles per second.[3] Heart sounds were recorded externally from a location just medial to the apex beat by either a Schwarzer crystal microphone and direct writing recorder or by a Groom dynagage capacitance microphone[4] and Midwestern photographic recorder. The medium frequency (MS) channel at 70 c.p.s. with a steepness of 18 db/octave of the Schwarzer recorder was used. From the Groom microphone, recordings were band-passed through a Spencer-Kennedy filter from 20 to 70 c.p.s. Tracings of heart sounds were similar from either recording system. Cardiac outputs were determined using indocyanine green.[5] Injections were made rapidly into a peripheral vein and samples withdrawn from the ascending aorta or left ventricle. An EKG was recorded throughout each study.

From these data the greatest amplitude of the first (S_1) and second heart sound (S_2) complexes were measured in millimeters. As an index of contractility, the maximum rate of pressure rise was measured graphically from the peak positive slope on the left ventricular pressure recording.[6] Isometric contraction time was measured from the beginning of the first sound to the onset of aortic pressure rise.

Baseline recordings before drug administration were compared to readings during peak drug effect. A stable state was permitted between phases of the study to allow for dissipation of effect from the previous drug. Approximately two hours was required for each study. Amyl nitrite was given first, followed by norepinephrine and lastly, methoxamine. The patient inhaled from a broken ampule of amyl nitrite containing five minims. Norepinephrine bitartrate (conc. of 4 mgm/ml) was infused at 40-60 drops per minute. Methoxamine hydrochloride (1 mg/ml) was infused at a rate of 2 ml/minute. The averages of ten sequential heart beats for

TABLE 1

TYPICAL RESPONSE TO DRUGS

	Amyl Nitrite	*Methoxamine*	*Norepinephrine*
First Sound	increase	decrease	increase
Second Sound	decrease	increase	increase
Cardiac Output	increase	decrease	same
(Contractility	same	same	increase)
*Systemic Blood Pressures	decrease	increase	increase
Total Peripheral Resistance	decrease	increase	increase
Heart Rate	increase	decrease	decrease
Stroke Volume	same	increase	increase

There were no changes in P-R intervals, isometric contraction times nor end diastolic pressures of the left ventricle.
*Includes systolic, diastolic, dicrotic and mean arterial levels, all of which changed similarly.

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TABLE 2

MEAN CHANGES AND STANDARD DEVIATIONS FOR THREE DRUGS

	Base	*Amyl Nitrite*	*Base*	*Methoxamine*	*Base*	*Norepinephrine*
S_1 in mm.	14.7 ± 5.9	25.7 ± 12.6	16.5 ± 7.8	12.4 ± 6.9	10.2 ± 4.7	17.9 ± 8.9
S_2 in mm.	11.9 ± 8.5	5.4 ± 4.1	9.0 ± 5.9	16.4 ± 7.0	8.2 ± 4.5	13.5 ± 7.3
Cardiac Output L/Min	5.4 ± 2.4	7.4 ± 3.3	5.2 ± 2.0	3.8 ± 1.6	4.8 ± 1.3	4.8 ± 1.7
T.P.R. dynes.sec.cm.$^{-5}$	1702 ± 648	830 ± 374	1671 ± 607	3270 ± 1817	1787 ± 585	2603 ± 1169
Mean BP mm. Hg.	102 ± 19	73 ± 25	97 ± 20	127 ± 25	105 ± 21	125 ± 33
Dicrotic BP mm. Hg.	88 ± 23	60 ± 24	88 ± 23	126 ± 31	94 ± 37	147 ± 32
Systolic BP mm. Hg.	122 ± 22	83 ± 18	117 ± 22	159 ± 36	126 ± 25	164 ± 62
Diastolic BP mm. Hg.	78 ± 16	51 ± 22	76 ± 16	93 ± 16	80 ± 16	99 ± 20
Heart Rate per min.	80 ± 14	112 ± 22	81 ± 14	57 ± 20	74 ± 17	59 ± 11
Stroke Volume cc.	68 ± 24	67 ± 28	63 ± 27	74 ± 24	69 ± 13	82 ± 23

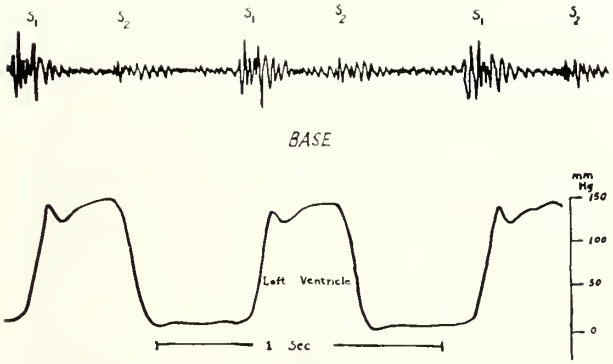


Fig. 1

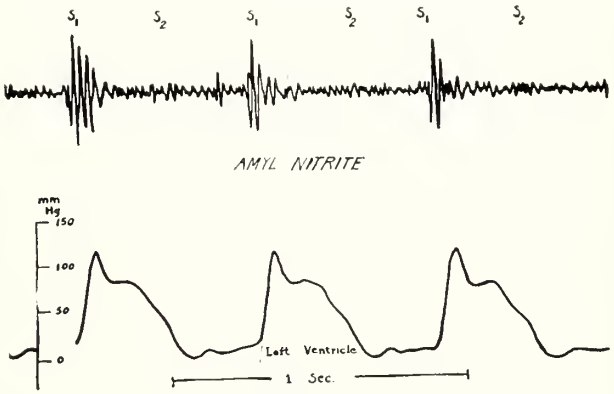


Fig. 2

each value measured were determined. Two baseline cardiac outputs at the beginning of each experiment were compared to one cardiac output taken during the peak effect of drug administration.

RESULTS

Typical responses to each of the drugs are summarized in Table 1. The changes in hemodynamics are similar to those of previously published studies.[7–11] When compared with numerical values for baselines, all changes including those of heart sounds were statistically significant ($P > 0.05$). The response of contractility (Table 1) is placed in parenthesis because there were not enough technically satisfactory measurements of maximum rates of rise in left ventricle for statistical analysis. The direction of change in contractility is, however, the same as reported by others.[8,9,11] None of the drugs produced changes in P-R intervals, isometric contraction time nor end diastolic pressures of the left ventricle.

Numerical values measured are appended in Table 2 indicating mean changes and standard deviations for each of the drugs.

AMYL NITRITE, (Figs. 1 and 2). The typical response, and it was rather constant, was a marked increase in S_1 and a marked decrease in S_2. Heart rate increased; blood pressure fell. Cardiac output increased to an average of 43% above base level. The contour of the arterial pressure was changed; there being a lowering of the dicrotic notch to diastolic levels or below.

METHOXAMINE. (Figs. 3 and 4). The usual response

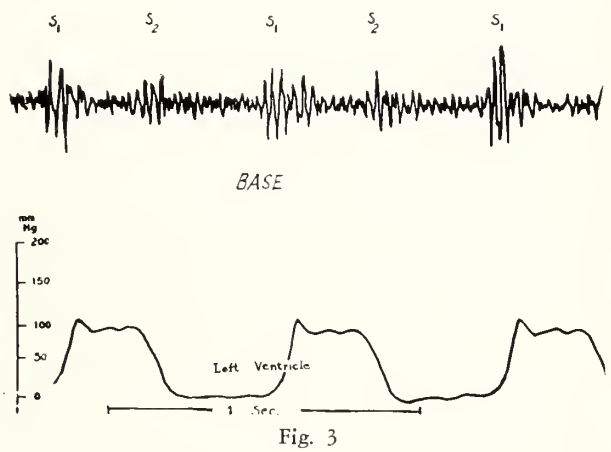


Fig. 3

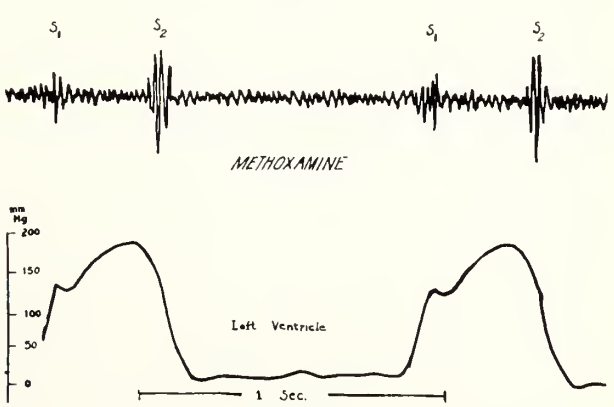


Fig. 4

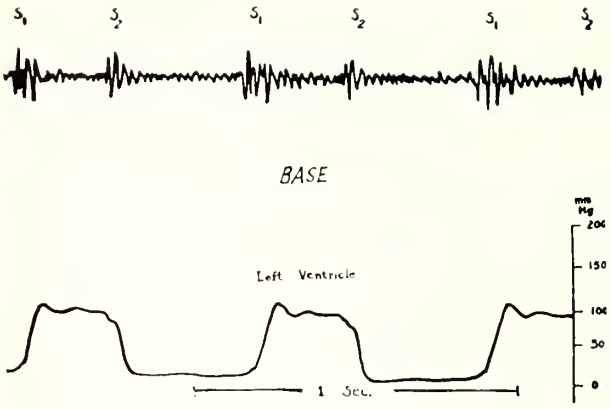


Fig. 5

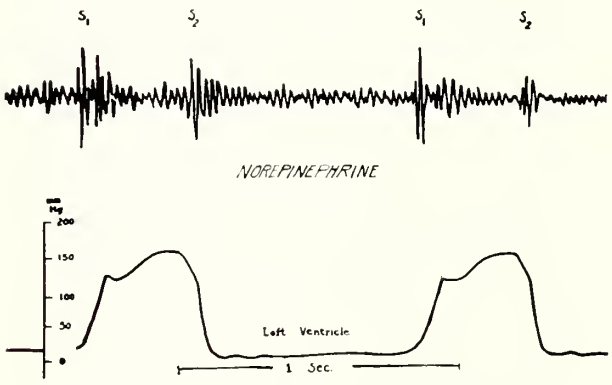


Fig. 6

in hemodynamics and heart sounds was opposite to, although not as striking nor consistent as, that with amyl nitrite. The first heart sound diminished and the second increased. Blood pressures rose markedly, usually to levels of above 200 systolic and 100 diastolic. Heart rate slowed to an average of 57 beats per minute. Cardiac output diminished, averaging a reduction of 26%. Stroke volume average increased from 63 to 74 cc. The arterial pressure curve developed an anacrotic shoulder and the dicrotic notch moved higher on the descending limb.

NOREPINEPHRINE, (Figs. 5 and 6). A distinct increase in amplitude of both first and second heart sounds was noted. Systolic and diastolic blood pressures rose. An increase in maximal rate of rise of pressure in the left ventricle (average +82%) was noted in each of four patients in whom determinations were obtained. Heart rate slowed and stroke volume increased from an average of 63 to 83 ccs. per beat. The pressure pulse developed an anacrotic shoulder and elevation of the dicrotic notch.

DISCUSSION

From external heart sound recordings, the supposed contributions from mitral or tricuspid valve components cannot be separated. However, the factors in this study that changed the amplitude of the first heart sound affected both ventricles similarly. Because it closely preceded the dicrotic notch of aortic tracings, the second sound is presumed to relate primarily to aortic valve closure.[12]

FIRST HEART SOUND: With amyl nitrite, an increase in cardiac output and increase in rate were accompanied by an increased intensity of the first heart sound. When methoxamine was given, both the output and rate decreased as did the first heart sound's intensity. With norepinephrine, the rate decreased but the heart sound intensity increased. No change in cardiac output occurred yet there was an increase in the first heart sound's intensity. A cause of this increase is perhaps the increase in myocardial contractility with norepinephrine. This factor did not change, however, with the other two drugs in which there were changes in output. Increases in peripheral resistance were accompanied in one instance by an increase in the sound (norepinephrine) and by a decrease (methoxamine) in the other. With amyl nitrite, the intensity was increased while peripheral resistance was decreased. With the increase in heart rate accompanying amyl nitrite, the first sound intensity increased but likewise increased accompanying the bradycardia from norepinephrine. Similarly stroke volume increase was associated with a decrease in heart sound intensity with methoxamine and with an increase with norepinephrine.

It would appear that changes in cardiac output and contractility could be profitably studied for a more exact delineation of their apparent influence on the intensity of the first heart sound. One animal study has, in fact, already related these changes to contractility.[13] Unfortunately, cardiac outputs were not determined in the animals. It would be a useful bedside guide to the clinician if he knew that increased intensity of the first heart sound reflected improved contractility of the heart or improved cardiac output. It is possible that it would aid him in titrating digitalis therapy.

The previously well established relationship between first heart sound intensity and P-R interval[14–15] was confirmed by us in ancillary observations of the effects of carotid sinus stimulation. The effects of the drugs, however, were more dominant. Also, in our one example of atrial fibrillation, the expected variation in heart sound intensity[16] was "over-ridden" by the drug effect.

SECOND HEART SOUND: When systemic blood pressures and peripheral resistances were altered by the drugs, similar alterations occurred in the intensity of the second sound. Opposite changes occurred with alterations of heart rate. With both methoxamine and norepinephrine, increases in stroke volume were associated with increases in the second heart sound, but its intensity was diminished with amyl nitrite which did not change stroke volume. Similarly as cardiac output changed with amyl nitrite and methoxamine, an opposite change occurred in intensity of the second heart sound, but when output did not change with norepinephrine, its intensity was markedly increased. Changes in the heart sound occurred with amyl nitrite and methoxamine unaccompanied by changes in contractility.

Observations on the intensity of the second heart sound with carotid sinus stimulation tend to rule out a rate effect, which might be implied from the above. The most promising area for further exploration would be the relationship to systemic pressures. Clinically this has been familiar in the accentuated aortic sound of hypertension. Cardiac output seems from the above to be not related to second heart sound intensity and the relationship between intensity and total peripheral resistance (because the calculation of the latter involves output) would be expected to be less than that to systemic pressures. This was found to be the case when correlation factors were calculated. Further, the calculation of correlation factors indicated that the systolic pressure related less to the second sound intensity than the diastolic pressure. The number, however, yielding the best correlation was that of centrally determined aortic dicrotic level. This level reflects the closure of the aortic valve. An observation of Kjellberg[17] supports our minimizing the role of systolic pressure; when adrenalin was given he noted no change in the second heart sound when systolic pressure alone was elevated.

Summary

Alterations in heart sounds and hemodynamics were produced by amyl nitrite, methoxamine and norepinephrine. Further study of the hemodynamic factors responsible for the changes is indicated. Inferences, which may perhaps be of some clinical usefulness have, however, been noted.

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CARDIOPULMONARY RESUSCITATION AT A COMMUNITY TEACHING HOSPITAL — Report of 37 Cases

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"'Tranquilizer' is not a good word"[1]

"THIS classification is psychologically too seductive, pharmacologically too unspecific, and in terms of results not infrequently untrue."[2]

What is a tranquilizer? According to the 24th Edition of Dorland's Medical Dictionary[3] a tranquilizer is "an agent which acts on the emotional state, quieting or calming the patient without affecting clarity of consciousness."

Defining a drug by its effects, however, can be misleading. The same effects by which the dictionary defines a tranquilizer have sometimes been seen after administration of a sedative — or, for that matter, a placebo.

Ambiguous though the term may be, it appears to be here to stay. The 1966 edition of the Physicians' Desk Reference[4] lists 42 tranquilizers indicated for treatment of anxiety and apprehensive states.

'Tranquilizers' have differences in action, differences in effect

Although all tranquilizers are intended to calm anxious patients there *are* differences in their actions — and in their effects. They have been divided into three categories — the rauwolfia group, the 'minor' tranquilizers, and the phenothiazines.[5]

Although the tranquilizing effect of rauwolfia has been known for centuries, its use as an antipsychotic agent in current practice has diminished.[5]

A 'minor' tranquilizer is often prescribed to achieve more than one effect. Thus, besides being tranquilizers some of these compounds may be muscle relaxants, antihistaminics with some calming action, anticholinergic sedatives, or antispasmodics.[5]

The phenothiazines are considered 'major' tranquilizers because they alter psychotic behavior.[1] This classification may have done them more harm than good because it implies that the phenothiazines should be reserved for the more severely disturbed. This is not necessarily true.

The phenothiazines — and the problem of sedation

One of the problems of prescribing phenothiazines for ambulatory patients has been the fear that excessive sedation will impair the patient's ability to function. This, however, is less of a problem with some of the phenothiazines.

"Clinically they may be differentiated primarily in terms of their potency and the extent of their sedative effect, which appear to be inversely proportional. That is, the least potent, the one which is used in highest dosage, chlorpromazine, is the most sedative, while the reverse holds true for fluphenazine."[6]

In a recent report on various studies conducted over several years evaluating 360 patients treated for anxiety and stress with seven phenothiazines, this inverse relationship of potency to sedation was confirmed.[7] Also under consideration was the degree to which the particular phenothiazines neutralized anxiety (the angolytic index). Interestingly enough there was, again, an inverse relationship. The most sedative of the phenothiazines appeared to be the least active in neutralizing anxiety. Fluphenazine, one of the least sedative, on the other hand, was found to be most effective in relieving anxiety.[7]

RELATIVE SEDATIVE AND ANGOLYTIC INDICES OF PRINCIPAL PHENOTHIAZINES*

DRUG	SEDATIVE INDEX	ANGOLYTIC INDEX	BASED ON STANDARD DOSE OF
Chlorpromazine	100	15	25 mgs.
Triflupromazine	100	15	25 mgs.
Thioridazine	90	17	25 mgs.
Perphenazine	15	25	4 mgs.
Carphenazine	25	25	25 mgs.
Trifluoperazine	3.3	95	2.0 mgs.
Fluphenazine	3.5	100	2.5 mgs.

*adapted from Sainz[7]

Prolixin is therapeutically effective without excessive sedation

When used as a 'tranquilizer' in general medical practice, in many patients Prolixin (Squibb Fluphenazine Hydrochloride) suppresses anxiety, but not normal activity. These two features are of particular importance to patients who must be able to live and work without their normal daily activities being restricted.

Because of its longer duration of action, Prolixin, in doses of as little as one to three milligrams in adults, generally taken once a day, is effective in maintaining many patients free of their symptoms of anxiety and tension.

Contraindications: Do not use with high doses of hypnotics or in patients with subcortical brain damage. Use with caution in patients with a history of convulsive disorders. Severe reactions may occur in patients with idiosyncrasy to other centrally-acting drugs, and severe hypotension may occur in patients with special medical disorders, e.g. mitral insufficiency and pheochromocytoma.

Precautions: Effects of atropine, anesthetics and C.N.S. depressants may be potentiated. Hypotension may occur in patients undergoing surgery. Do not use epinephrine for treatment of the hypotensive reactions which may appear in patients on phenothiazine therapy.

Side Effects: Extrapyramidal reactions, allergic skin reactions, the possibility of anaphylaxis, drowsiness, visual blurring, dizziness, insomnia, nausea, anorexia, salivation, edema, perspiration, dry mouth, abnormal lactation, polyuria, hypotension, and jaundice and biliary stasis may occur. Routine blood counts are recommended to determine possible blood dyscrasias; if symptoms of upper respiratory infection occur, discontinue drug and institute appropriate therapy.

Available: 1 mg. tablets. Bottles of 50 and 500.

For full prescribing information, see package insert

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DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Maine's Expanded Medical Care Program—Title XIX

Date: June 30, 1966

To: Administrators of all Hospitals Certified Under Medicare

From: Dean Fisher, M.D., Commissioner, Department of Health and Welfare, Augusta

Subject: Provision of Hospital Care and Related Services by the Department of Health and Welfare for all Public Assistance Recipients (Old Age Assistance, Aid to Families with Dependent Children, Aid to Disabled, Aid to Blind) under Titles 18 and 19 of Medicare

This memorandum describes the Department's services, policies, procedures and methods of payment, effective July 1, 1966, in relation to the hospital and related care aspects of its new Medical Assistance program provided under Medicare. This program will supersede all other medical care programs for this group of recipients. You will recall our earlier notification regarding the termination of our M.A.A. program effective July 1, 1966, which will be replaced for most M.A.A. recipients by the Medicare services, provided appropriate applications for Medicare were made. State Hospital Aid, as partial assistance to the medically indigent under 65 years, other than public assistance recipients, will continue at a reduced per diem payment of $5. The reason for this reduction in State Hospital Aid is primarily due to the fact that every $1 of State funds used in our other programs generates about $2 of Federal money; we can, therefore, more adequately reimburse hospitals for care provided public assistance recipients if we use part of our State Hospital Aid funds to generate Federal dollars. This simply means that rather than the $1 hospitals have received from State Hospital Aid, hospitals will now receive $3 in public assistance welfare medical payments.

I regret that Medicare timetables and Federal directives did not permit joint meetings between hospital personnel and the Department's staff, but I hope that this new medical assistance program to be provided under the Medicare law and regulations is sufficiently spelled out in this memorandum for implementation by the hospitals without undue complications on July 1, 1966.

We are aware, however, of the possible omission of unforeseeable questions and problems, and will welcome your inquiries to our Medical Unit in Augusta, – Mr. Robert Wyllie, 623-4511, extension 304.

In addition to this memorandum to hospitals, we are sending communications to all physicians, and other providers of services under Medicare as well as to the public assistance recipients. (A copy of the letters sent to physicians was enclosed with material sent to hospital administrators for their information).

I. *Recipients over 65 years of age (Old Age Assistance)*

A. *Coverage:* Our Old Age recipients will be eligible for all of the benefits listed under Parts A & B of title 18 of Medicare plus the following additional services:

1. Necessary routine physical examinations.
2. Immunizations.
3. All outpatient services.
4. First three pints of blood.
5. Hospitalization beyond 90 days for each "spell of illness", if necessary.
6. Home Health services beyond 200 visits if medically necessary.
7. Hospital coverage beyond 90 days if necessary in any one "spell of illness".

The State will pay for additional inpatient hospitalization when the request with adequate medical information is presented in writing to the Department at least 10 days prior to the end of the 90 day period. Justification for continuing payment of inpatient hospital care beyond 90 days will then be required every 14 days in writing to the Department. This report must contain adequate up-to-date information as to diagnosis, complications, prognosis and a definite plan for future patient care. A copy of the hospital's utilization committee evaluation of the need for hospitalization may be substituted for the report if appropriate and if it includes the above information.

Billing for above 7 items of extended coverage will be through the fiscal intermediaries (Associated Hospital Services or the Union Mutual Insurance Co.).

B. *Methods of Payment*

1. *For Part A. of Medicare* (Hospital Payments) –

the Department will use the fiscal intermediary (Associated Hospital Services) for payment of all claims on behalf of its Old Age Assistance recipients. All expenses incurred through required deductibles ($40.00) co-insurance ($10.00 per day from the 60th to 90th day for a spell of illness) plus extended benefits will be covered. Thus inpatient services and outpatient services provided by the hospital will be paid for through the fiscal intermediary as will those services provided under Part B which are also provided by the hospital.

2. *For Part B of Medicare* – the State will provide payment for service to its Old Age Assistance recipients through the Union Mutual Insurance Company. All physicians' services under Part B plus extended services noted above as well as ($50) required deductible and 20% co-insurances payments will be covered.

These arrangements have been made to coordinate the State's program for its Old Age Assistance recipients with all Medicare beneficiaries age 65 and over. It means that all hospitals will be dealing directly with the Associated Hospital Services of Maine for all individuals in this age group. Please send your bills to the fiscal intermediary for our Old Age recipients in exactly the same manner as for all individuals age 65 and over. Actual hospital costs, deductibles, co-insurance and payment for extended coverage will be paid to you directly (from the AHS) for our recipients. This program for payment also means that the rates of payments must be accepted as payment in full, minus any individual insurance payments and voluntary payments by the patient, his family or other sources.

In order to facilitate processing of these claims the hospitals must identify on Medicare Form SSA-1453 – Inpatient Hospital Admission and Billings, under Section 12, that the patient is an Old Age Assistance recipient. The fiscal intermediary will have current information indicating the recipient's eligibility for payment. A report of admission to the hospital with a preliminary or working diagnosis must be forwarded to the Department of Health and Welfare on the present G-47 form at the time of admission.

C. *Hospital Based Specialists* – the administrative services of these specialists to the hospital, if any, will be included in the operating costs under Part A of Medicare, and processing therefore will be through the Associated Hospital Services in accordance with their methods of payment.

The services of these specialists, directly related to diagnosis and treatment are covered under Part B of title 18, and payment should be processed through the Union Mutual Insurance Co., in accordance with their methods of payment regardless of individual assignment arrangements between the hospitals and specialists.

D. *Certification of Need for Care* – will be handled in exactly the same way as for all title 18 beneficiaries.

E. *Utilization Review* – will be handled in exactly the same way as for all medicare beneficiaries. However, the Department retains the right to ask for copies of the utilization committees reports on any patient in a hospital for over 30 days.

II. *Public Assistance Recipients under 65 years of Age (Aid to Families with Dependent Children, Aid to the Disabled, and Aid to the Blind).*

The medical assistance program for these recipients is covered by the law and regulations of title 19 of PL 89-97. The program will be administered directly by the Department of Health and Welfare and will not go through the two fiscal intermediaries as described above for those recipients age 65 and over.

These recipients under age 65 will carry initially a letter of identification and by the end of July 1966 an identification card valid for 6 months. However, because of the unpredictable factors in continued public assistance, the hospital cannot assume current eligibility and should protect itself by forwarding to the Department, upon admission of the patient, the usual G-47 for verification of eligibility. Notification will be returned to the hospital as expeditiously as possible.

A. *Coverage and Scope:* All public assistance recipients in the categories noted above will be eligible for all of the benefits listed under Parts A & B of title 18 of Medicare plus the following additional services:

1. Necessary routine physical examinations.
2. Immunizations.
3. All outpatient services.
4. First three pints of blood.
5. Hospital coverage beyond 90 days if necessary.
6. Home health services beyond 200 visits if medically necessary.

The State will pay for additional inpatient hospitalization when the request with adequate medical information is presented in writing to the Department at least 10 days prior to the end of the 90 day period. Justification for payment of inpatient hospital care beyond 90 days will then be required every 14 days in writing to the Department. This report must contain adequate up-to-date information as to diagnosis, complications, prognosis and a definite plan for future patient care. A copy of the hospital's utilization committee evaluation of the need for hospitalization may be substituted for the report if appropriate and if it includes the above information.

B. *Payment* – The Department of Health and Welfare will act directly as the fiscal agent for these recipients. The same rates and basis of payment will be used as by the two fiscal intermediaries for Parts A & B of title XVIII of Medicare.

For the present, the Department's established reporting and billing forms will be used, G-47 for admission and discharge reporting and Form A & A-6 for billing. A new G-47 form and new billing form for inpatient services will be developed in the future and you will then receive a supply.

Reporting and billing of outpatient services and of medical and other health services provided by the hospital will be on a special outpatient form. Temporary

billing forms for this purpose will be forwarded to you within 10 days. This form will be similar to those used by the fiscal intermediaries for Parts A & B (Title XVIII).

The Department would appreciate the hospital's efforts to keep current with billings in this expanded program with which the Department has had no expenditure experience.

It should be noted that *medicine* other than that provided during inpatient care, or that necessarily administered by a physician on a non-in-patient basis, as a part of his services, are not covered by either titles 18 or 19 of PL 89-97.

C. *Hospital based Specialists* – The Department will follow the same methods of payment and policies for title 19 reimbursements as are used by the fiscal intermediaries for title XVIII except that, under title 19, there should be vendor payments only.

Payment by the Department in accordance with the above rates must be accepted as payment in full minus any deductions for individual insurance payments, and voluntary payments by the patient, family or other sources.

D. *Certification of need for Care* – Necessary on in-patients only and will be handled in exactly the same way as for all title 18 beneficiaries.

E. *Utilization Review* – The Department will require that the same utilization review plan be carried out by each hospital for these public assistance recipients under age 65 as for those Medicare beneficiaries age 65 and over. The Department retains the right to ask for copies of the utilization committees reports on any patient in a hospital for over 30 days.

III. *Community Resources for Discharge Planning*

As a possible help to the hospitals' administrative, medical, nursing and social work staffs in discharge planning and in medically feasible alternate planning in place of extended hospital care, we will keep you informed of certified home health agencies in your hospital area as they become certified. This list will be currently limited; it is anticipated that others will soon be certified. Each hospital will be familiar with the home health care or visiting nurse agencies in its community, whether or not certified, in addition to the services of the State Division of Public Health Nursing. Certification under Medicare means that in addition to meeting other Conditions of Participation, and the provision of nursing service in the home, also included are some consultation and direct services by one of the following: physical therapist, occupational therapist, medical social worker, speech therapist, and home health aide.

Home Health Services – The Department will pay for unlimited Home Health Services for all public assistance recipients in all age groups. However, billing for the under age 65 group will be directed to the Department of Health and Welfare and for the 65 and over age group to the fiscal intermediaries for Part A, title 18. Utilization of services from certified agencies must be similar for those under age 65 as for the persons 65 and over.

Also in relation to discharge planning, the Department will provide to the hospitals within the next several months information on certified extended care facilities, personal and boarding care facilities.

IV. It should be clearly stated again that when reference is made above to "Public Assistance Recipients", the term refers to only the recipient of Old Age Assistance, Aid to the Disabled, and Aid to the Blind and not to the spouse. The spouse will not be eligible for these medical care services unless he is a recipient of a public assistance grant himself.

In the instance of an AFDC family, each child for whom an AFDC payment is made is eligible for medical care; the parent receiving AFDC assistance will also be eligible and if there are two AFDC parents included in the grant, both will be eligible for medical care payments.

Date: July 20, 1966
To: Administrators of Hospitals Certified for Medicare Reimbursement
From: Dean Fisher, M.D., Commissioner
Subject: Revised G-47 Form and Billing Information – TITLE XIX

I. *Revised G-47*

The revised G-47 form (copies were enclosed with this letter) serves as a notification of admission, a discharge notice and as a billing form. Again, we recognize that these are temporary forms but to develop a form similar to those used for TITLE XVIII, it was necessary to wait until such was available to us; The formal printed billing forms will be slip-sheeted with carbons for your convenience.

Upon admission of a public assistance recipient under age 65 you will complete the first part of the entire set of forms, tear off sheet #4 (Notification of Admission) and forward it to the Division of Accounts and Audit, Department of Health & Welfare, State House, Augusta, Maine. Eligibility status of the person will be determined, noted on the form, and returned to you for billing purposes.

At the time of discharge additional information requested should be entered on sheet #3 and forwarded as above. If billing accompanies the discharge notification, all three copies can be submitted together. *You need no longer forward a G-47 to this Department upon the admission of a public assistance recipient age 65*

Continued on Page 292



following infection

B and C vitamins are therapy: STRESSCAPS B and C vitamins in therapeutic amounts... help the body mobilize defenses during convalescence... aid response to primary therapy. The patient with a severe infection, and many others undergoing physiologic stress, may benefit from STRESSCAPS capsules.



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Each capsule contains:

Vitamin B_1 (as Thiamine Mononitrate)	10 mg
Vitamin B_2 (Riboflavin)	10 mg
Vitamin B_6 (Pyridoxine HCl)	2 mg
Vitamin B_{12} Crystalline	4 mcgm
Vitamin C (Ascorbic Acid)	300 mg
Niacinamide	100 mg
Calcium Pantothenate	20 mg

Recommended intake: Adults, 1 capsule daily, for the treatment of vitamin deficiencies. Supplied in decorative "reminder" jars of 30 and 100; bottles of 500.

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Maine Heart Association Notes

116 State Street, Augusta, Maine

Selective Revascularization of the Myocardium by Internal-Mammary-Artery Implant

Twenty-four months' experience with internal-mammary-artery surgery for relief of angina pectoris and myocardial ischemia justifies cautious optimism. There is sound morphologic and physiologic evidence that the operation revascularizes a portion of the human heart. . . . Ideally, the patient with disease confined solely to the left coronary arteries should benefit most from this operation. However, since the patient with 3-vessel disease so obviously needs help, surgery should not categorically be denied him simply because of the increased operative risk and the fact that the implant may not adequately revascularize the inferior and posterior zones of the heart.

. . . patients with minor disease clearly should be rejected for surgery.

The intrinsically high mortality of the disease will remain the major factor militating against a low operative mortality (5%).

Major considerations pertinent to intraoperative success are hemodynamic and respiratory support of the patient. . . .

Persistent relief of angina pectoris occurred in approximately 80% of patients. This . . . agrees with the reported incident or of patency of the internal mammary artery.

Forty patients . . . underwent implantation of an internal-mammary-artery pedicle.

Only the patients with severe angina pectoris or those with a history of repeated myocardial infarctions under the age of forty-five years were accepted as candidates for surgery.

There were two hospital deaths and two late deaths during this first two-year period of observation.

Of 26 patients evaluated seven to thirty-four months after surgery 21 were subjectively improved.

Morphologic patency of the implant was documented by selective cinearteriography in 11 of 13 patients studied one year after operation.

Submitted by Jacob B. Dana, M.D., Maine Heart Association.

Reference—Gorlin, R. and Taylor, W., The New England Journal of Medicine, Vol. 275, pages 283-290, 1966.

Most of my patients with high blood pressure are as old as I am. A lot of them are living on pensions. They're grateful when I can keep prescription costs down.



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chlorthalidone 50 mg. reserpine 0.25 mg.

1 tablet daily brings pressure down

Advantage: Both components of Regroton are long-acting.
Average dosage: One tablet daily with breakfast.
Contraindications: History of mental depression, hypersensitivity, and most cases of severe renal or hepatic diseases.
Warning: Discontinue 2 weeks before general anesthesia, 1 week before electroshock therapy, and if depression or peptic ulcer occurs. With administration of enteric-coated potassium supplements, the possibility of small bowel lesions should be kept in mind.
Precautions: Reduce dosage of concomitant antihypertensive agents by one-half. Discontinue if the BUN rises or liver dysfunction is aggravated. Electrolyte imbalance and potassium depletion may occur; take particular care in cirrhosis or severe ischemic heart disease, and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended. Use with caution in patients with ulcerative colitis, gallstones, or bronchial asthma.
Side effects: Nausea, vomiting, diarrhea, muscle cramps, headaches and dizziness. Potential side effects include angina pectoris, anxiety, depression, drowsiness, hyperglycemia, hyperuricemia, lassitude, leukopenia, nasal stuffiness, nightmare, purpura, urticaria, and weakness.
For full details, see the complete prescribing information.
Availability: Bottles of 100 and 1000 tablets.

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AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Hydrogen Bonding May Give Clue to Mechanism of Mutation

Scientists at Massachusetts Institute of Technology in Cambridge, Massachusetts have shown that one of the major consequences of a gene mutation may be an alteration in the hydrogen bonds which hold two strands of the DNA, or gene, molecule together.

Inasmuch as most authorities feel that cancer usually or always is a result of the mutation of a body cell gene, the finding may help explain the chemistry of cancer.

This research under the direction of Dr. Alexander Rich was reported recently by the American Cancer Society. Associated with Dr. Rich in the studies were Drs. F. Scott Mathews, Louis Katz and Ken-ichi Tomita.

Genes are made of DNA (deoxyribonucleic acid), a very long, segmented, two-stranded molecule. The segments contain bases which are indicated by their initials, A, T, G and C (adenine, thymine, guanine and cytosine); each is attached to a sugar which is connected to its neighboring segments by a phosphate.

On the double strands of DNA, A is always paired with T and G with C. The strands are bound together by the attractive force of the hydrogen atoms in each strand segment.

Dr. Rich and his associates found that it was possible to form crystals of materials related to the A-T pair of the double stranded DNA.

One of the pairs involved A with U (uracil) which is closely related to the T base.

The other crystal involved A with a bromo U (bromouracil).

The molecules, U, bromo U and T are all very similar to each other. Bromo U is a completely artificial molecule and does not exist in nature. It differs from U in that a bromine atom is substituted for a hydrogen atom and it is very similar to T. The significant thing is the fact that bromo U is a mutagen which is known to bring about an alteration in the genes of experimental animals.

M.I.T. has made three-dimensional x-ray diffraction "pictures" of the A-U and A-bromo U complexes. These complexes are analogous to individual segments of a DNA molecule.

During these studies the group found that the replacement of the hydrogen atom by a bromine atom brought about a profound change in the way the two molecules were packed together in the crystal. This change in "hydrogen bonding" is reported to be related to the electrical forces within the molecule.

The fact that a known mutagen, bromo U, brings about a change in the hydrogen bonding may give a clue to the mechanism of mutation. This suggests that the alteration in the normal hydrogen bond which holds the DNA molecule together may be an important influence in altering genes. It may represent one of the mechanisms of mutation formation or cancer induction.

American Cancer Society News Service Release

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County Society Notes

LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held on September 20, 1966 at The Ledges in Wiscasset, Maine. The meeting was called to order by the President, Alan W. Zeller, M.D.

John F. Dougherty, M.D. asked that each physician express his appreciation of Maine Medical Association Journal advertising by writing to the advertisers. He mentioned the need of the Society for the Journal and its need for further revenue.

Dr. Dougherty stated that a communication from the U.S. Government has said that re-certification for extended in-hospital stay for Medicare patients need be written on no special form but as a progress note on the chart, signed by the physician.

Dr. Bostwick read a letter from the Industrial Accident Commission thanking the physicians for cooperating with scheduling of impartial medical examinations, and a letter from the Maine Medical Association outlining the procedure for reporting maternal mortality cases to the Committee to Conduct a Study of Maternal Mortality of the Maine Medical Association.

Niles L. Perkins, M.D., Director of the Bureau of Medical Care of the State of Maine Department of Health and Welfare, was introduced by the Secretary. Dr. Perkins spoke on the state program for medical care for indigent people.

The Lincoln-Sagadahoc County Medical Society met at The Ledges in Wiscasset, Maine on October 18, 1966. The President, Alan W. Zeller, M.D. called the meeting to order.

The guest speakers, Dr. Richard T. Chamberlin of Waterville and panel members, Drs. William B. Grow of Fairfield, Rudolph Haas and Louis N. Fishman of Lewiston representing the Maine Thoracic Society, were introduced by the Secretary. They spoke on pulmonary tuberculosis and on emphysema and respiratory failure.

GEORGE W. BOSTWICK, M.D., *Secretary*

KENNEBEC

The Kennebec County Medical Association met at the Senator Motel in Augusta, Maine on September 22, 1966. The meeting was called to order by the President, Allan J. Stinchfield, M.D.

A committee on resolutions was formed by the President in respect to the death of Adolphe J. Gingras, M.D. The committee consists of the following members: Drs. Giddings and Shelton, both of Augusta and Dr. Davis of Waterville.

A letter from Mr. Kendall H. Dunbar, Rehabilitation Counselor of the Industrial Accident Commission, was read. In the letter, Mr. Dunbar thanked the Maine Medical Association for their prompt and accurate attention to the forms required by the Industrial Accident Commission in cases of medical questions.

Alta Ashley, M.D. announced that Diabetes Week will be held during the month of November. She asked the Association for approval of the method of mass screening which has been proposed by the Diabetic Association. The State Laboratory will be obtaining capillary bloods and processing them through a recently acquired autoanalyzer. Considerable discussion followed and the members voted to go on record as approving the work of the Diabetic Association in this regard.

Drs. Robert L. Callahan of Augusta, Paul A. Marshall of Fairfield and Richard T. Chamberlin of Waterville, panel members of the clinical program, were introduced by Dr. Stinchfield. The panel discussed shock in its various forms, including some of the more recent developments in the field of diagnosis and follow-ups, such as central venous pressure and blood volumes

as well as in treatment including a discussion of the controversy over the use of vasopressors versus vasodilators.

RICHARD T. CHAMBERLIN, M.D., *Acting Secretary*

OXFORD

The Oxford County Medical Society held its annual meeting on October 5, 1966 at the Bethel Inn in Bethel, Maine.

The following officers were elected for 1966-1967:

President, Charles M. Smith, M.D., Dixfield
Vice-President, Harry L. Harper, M.D., South Paris
Secretary-Treasurer, Walter G. Dixon, M.D., Norway
Delegates to the Maine Medical Association House of Delegates: Drs. Peter B. Aucoin (1 yr.) and Linwood M. Rowe (2 yrs.), both of Rumford. Alternates: Drs. Joelle C. Hiebert, Jr. (1 yr.), Norway and Dexter E. Elsemore (2 yrs.), Dixfield

AKE AKERBERG, M.D., *Secretary*

HANCOCK

A meeting of the Hancock County Medical Society was held at the Brookside in Ellsworth, Maine on October 12, 1966.

Colonel William Law, U.S. Army (Ret.), Administrator of the Blue Hill Memorial Hospital, was the guest speaker. He spoke on the history of the U.S. involvement in Southeast Asia with particular emphasis on the geography, ethnic background and religion of the people in that area.

Guests present at the meeting in addition to Colonel Law were Drs. Randall Silver and Harry Fuller of Ellsworth, Maine.

RUSSELL G. WILLIAMSON, M.D., *Secretary*

New Members

HANCOCK

Nancy H. Stewart, M.D., 194 Main St., Bar Harbor
Winston G. Stewart, M.D., 194 Main St., Bar Harbor

OXFORD

Kevin Conway, M.D., Rumford Com. Hosp., Rumford
Hagop Halladjian, M.D., Rumford Com. Hosp., Rumford
Warren C. Hazelton, M.D., 2 E. Main St., South Paris

News and Notes

Three Maine Surgeons Cited by the American College of Surgeons

James L. Fife, M.D. of Brunswick, Clement A. Hiebert, M.D. of Portland and Carlton E. Swett, M.D. of Skowhegan were elected as Fellows of the American College of Surgeons on October 13, 1966 during the annual five-day Clinical Congress held in San Francisco, California.

The American College of Surgeons is a scientific, educational and voluntary association of surgeons, numbering 27,000 Fellows in 83 countries. The College was founded in 1913 to improve care of the surgical patient, and has pioneered in many directions in making surgical care as excellent as it is today.

DEPARTMENT OF HEALTH AND WELFARE – *Continued from Page* 285

and over. We have established a system with Associated Hospital Services to provide us with this information.

II. *Billing Information*

It has come to our attention that we alerted you to the fact that we would reimburse your facility on a "reasonable cost" basis, but neglected to define this for you specifically for billing purposes.

You should submit bills which reflect your regular charges and any subsequent computations to determine "reasonable cost" will be done in this office. We will reimburse your hospital for our recipients under age 65 on exactly the same basis and at the same percentage of charges as Associated Hospital Services will reimburse you for persons age 65 and over under TITLE XVIII of Medicare.

Furthermore, at whatever future date (for example – 6 months or 12 months) your hospital cost accounting is reviewed for purposes of checking TITLE XVIII reimbursement against actual costs for your respective facility, we also expect to have access to this information and to adjust our payment accordingly. At that time also if available data indicates you have been under-compensated for services given, an adjustment will be made retroactively on behalf of those public assistance recipients under age 65 whom you served. Conversely, if the review indicates that your facility has been over-compensated for services rendered we shall expect a reimbursement for that portion which constituted overpayment by our Department.

III. *Private Room*

We do not believe that established policies re: private rooms for public assistance recipients were clearly defined in our former memo. The following will outline those instances when this Department will reimburse for a private room.

1. *Public Assistance Recipients age 65 and over* – The policies established by the Social Security Administration for purposes of TITLE XVIII will apply equally to the person age 65 and over whether he is a recipient of public assistance or not. However, the Department will not pay the difference between the private room rate and the semi-private rate if the recipient elects same and it is not medically necessary.

2. *Public Assistance Recipients under the age of 65* – Reimbursement for a private room will be made by the Department only in those instances where such an arrangement is medically necessary. A form similar to the one used for TITLE XVIII which will substantiate such a necessity is in the process of development and a supply should reach you shortly.

In the event that a recipient under age 65 insists on, and is given, a private room when such is not medically necessary, the Department will not participate in the payment of *any part* of the room and board charge as long as the recipient remains in that room under those conditions. The Department will, however, participate in the payment of other covered services during the hospitalization.

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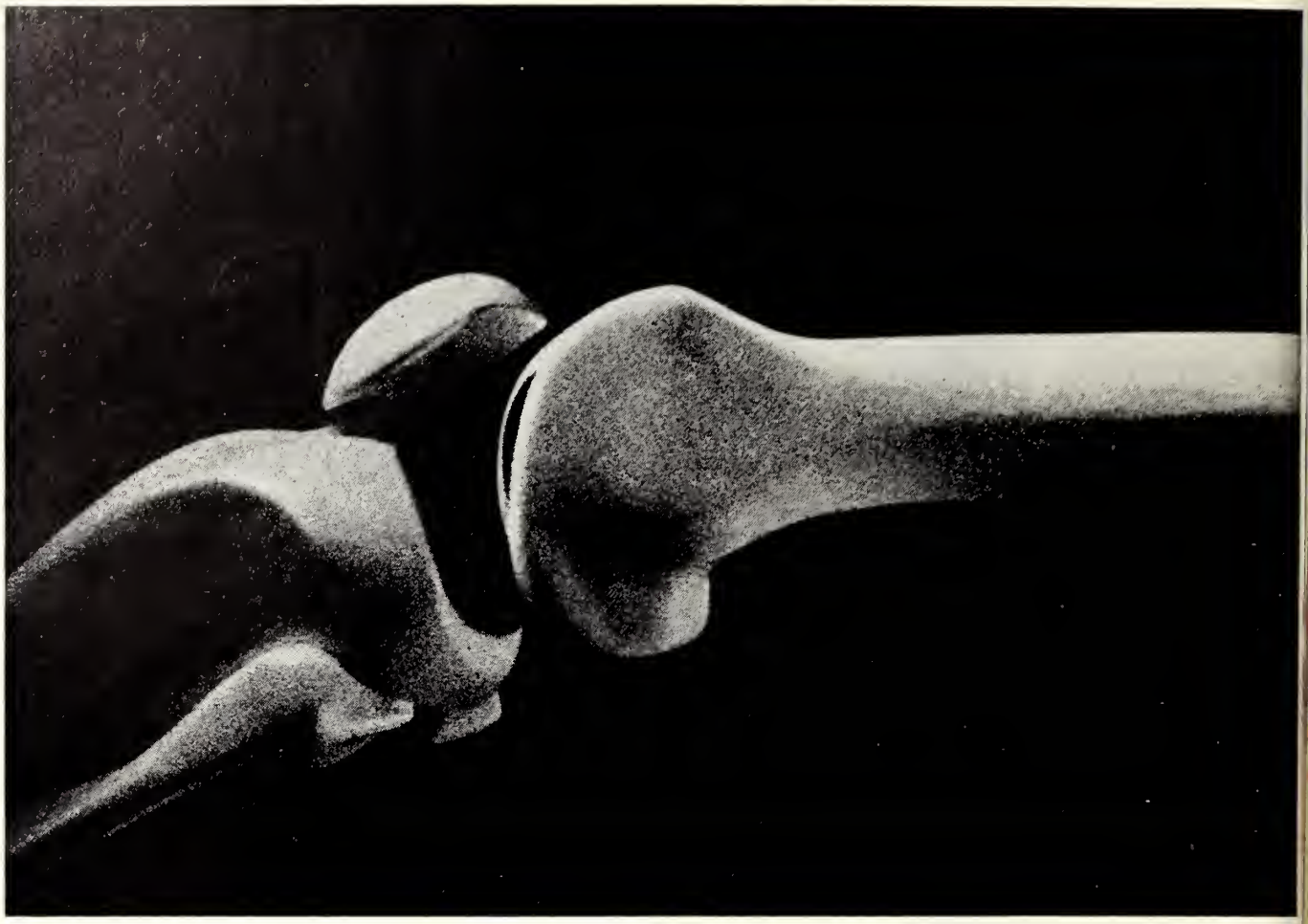
For your anxious patients who must remain active, 'Stelazine' offers a specific, incisive antianxiety effect that can calm excessive anxiety without producing annoying dulling effects. On 'Stelazine' patients react more normally to stresses and at the same time remain alert enough to carry on their normal activities.

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Before prescribing, the physician should be familiar with the complete prescribing information in SK&F literature or *PDR*. The following is a brief precautionary statement. *Contraindications:* Comatose or greatly depressed states due to C.N.S. depressants and in cases of existing blood dyscrasias, bone marrow depression and liver damage. *Precautions:* Use with caution in angina patients and in patients with impaired cardiovascular systems. Antiemetic effect may mask symptoms of other disorders. An additive depressant effect is possible when used with other C.N.S. depressants. Prolonged administration of high doses may result in accumulative effects with severe C.N.S. or vasomotor symptoms. Use in pregnant patients only when necessary for the patient's welfare. *Side Effects:* Occasional cases of mild drowsiness, dizziness, mild skin reactions, dry mouth, insomnia and amenorrhea. Neuromuscular (extrapyramidal) reactions (motor restlessness, dystonias, pseudo-parkinsonism) may occur and, in rare instances, may persist. In addition, muscular weakness, anorexia, rash, lactation, hypotension, and blurred vision have been observed. Blood dyscrasias and cholestatic jaundice have been extremely rare. For a comprehensive presentation of 'Stelazine' prescribing information and side effects reported with phenothiazine derivatives, please refer to SK&F literature or *PDR*.

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phenylbutazone, 100 mg.
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homatropine methylbromide, 1.25 mg.

Usually works within 3 to 4 days in osteoarthritis

The trial period need not exceed 1 week. In contrast, the recommended trial period for indomethacin is *at least 1 month.*

That's why it's logical to start therapy with Butazolidin alka—you'll know quickly whether or not it works. And usually, it will.

A large number of investigators have reported major improvement in about 75% of cases. Some patients have gone into remission. Relief of stiffness and pain may be followed quickly by improved function and resolution of other signs of inflammation. And Butazolidin alka is well tolerated, especially since it contains antacids and an antispasmodic to minimize gastric upset.

Contraindications
Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. Because of the increased possibility of toxic reactions, the drug should be used with greater care in the elderly and should not be given when the patient is senile or when other potent chemotherapeutic agents are given concurrently. Large doses of Butazolidin alka are contraindicated in patients with glaucoma.

Warning
If coumarin-type anticoagulants are given simultaneously, the physician should watch for excessive increase in prothrombin time. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Patients receiving such concomitant therapy should be carefully observed for this effect.

Use with caution in the first trimester of pregnancy.

Precautions
Before prescribing, the physician should obtain a detailed history and perform a complete physical and laboratory examination, including a blood count. The patient should be kept under close supervision and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools. Regular blood counts should be made to guard against blood dyscrasias.

Adverse Reactions
The most common adverse reactions are nausea, edema and drug rash. Moderately lowered red cell count may sometimes occur due to hemodilution. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, exfoliative dermatitis, Stevens-Johnson syndrome or a generalized allergic reaction may occur and require withdrawal of medication. Stomatitis, salivary gland enlargement, vertigo or languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia are also possible side effects.

Confusional states, hyperglycemia, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hepatitis, jaundice and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently.

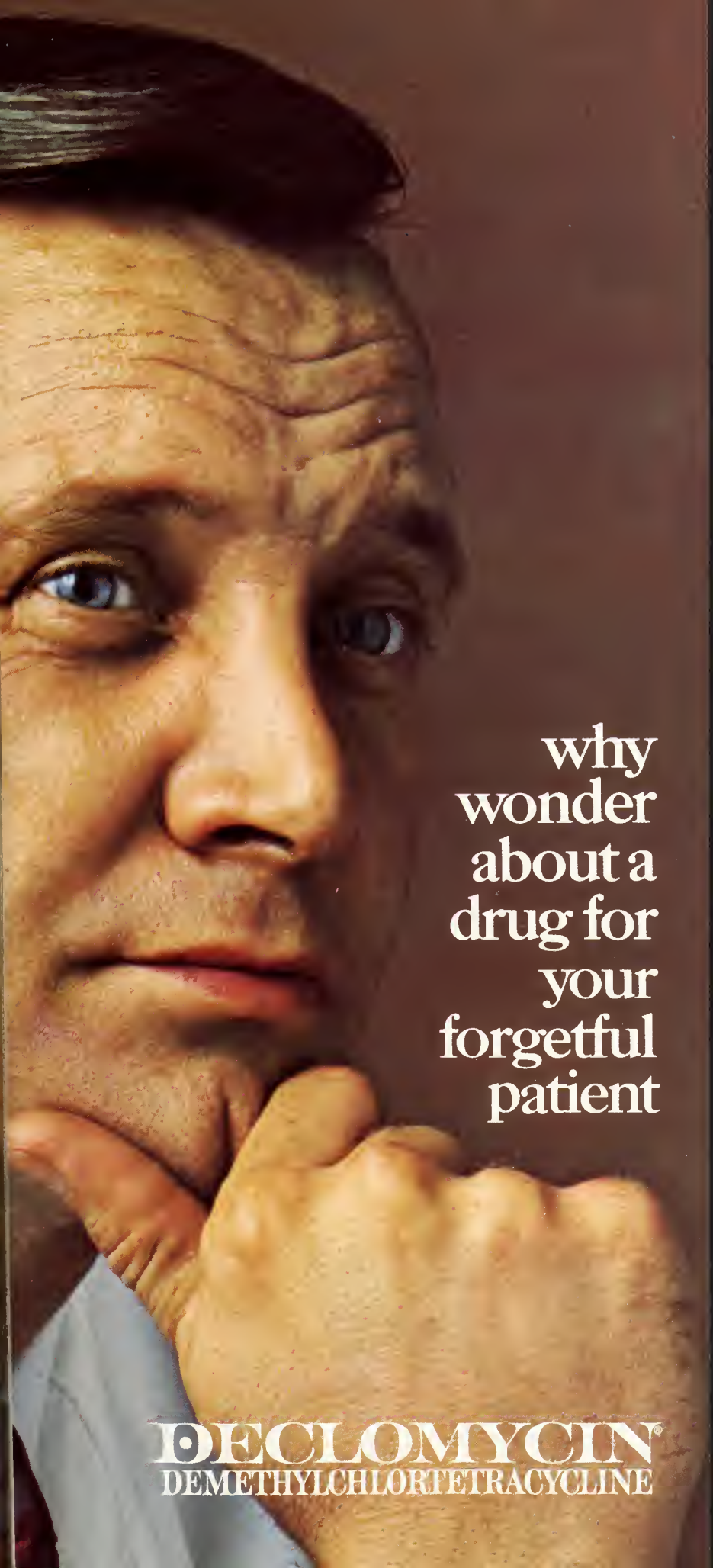
Dosage
The initial daily dosage in adults is 300-600 mg. daily in divided doses. In most instances, 400 mg. daily is sufficient. When improvement occurs, dosage should be decreased to the minimum effective level: this should not exceed 400 mg. daily, and is often achieved with only 100-200 mg. daily.

For complete details, please refer to full prescribing information.
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Effective in a wide range of everyday infections—respiratory, urinary tract and others—in the young and aged—the acutely or chronically ill—when the offending organisms are tetracycline-sensitive.

Contraindication—History of hypersensitivity to demethylchlortetracycline.

Warning—In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort.

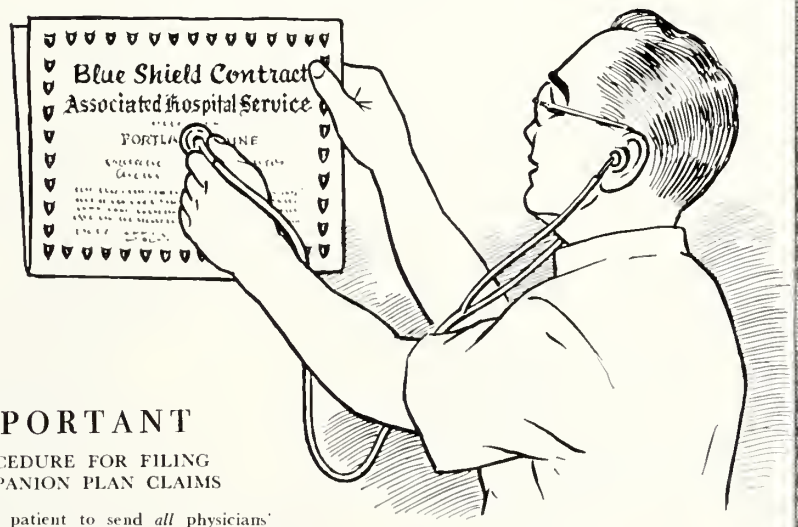
Precautions and Side Effects—Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

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IMPORTANT

PROCEDURE FOR FILING
COMPANION PLAN CLAIMS

Instruct your patient to send *all* physicians' bills to the A.H.S. office as received. Physician services must be itemized and their "reasonable charges" for each item indicated on the bills. A brief description of the injury or illness treated should also be shown. *The Companion Plan contract number and the Social Security or Government Identification number must be entered on the bill by the patient or the doctor.* For prompt direct payments of the Companion Plan benefits the bill should be issued to the patient in duplicate, one for Blue Cross-Blue Shield use and one for the Part B intermediary if you are not making claim on the Government Form SSA 1490.

If the bill is not receipted, payment will be made directly to the doctor on the regular semi-monthly Blue Shield check. The notation "C.P." for Companion Plan will be shown under the heading "Coverage" on the check voucher. This procedure will assure the doctor of the most prompt payment.

If the bill is receipted, payment of benefits will be made to the patient.

Medicare "Explanation of Benefits Form" may be used by the patient making claim for benefits but should be accompanied by itemized physicians' bills for all services incurred during the calendar year.

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Pleasant-tasting Alertonic is pipradrol hydrochloride—an effective cerebral stimulant whose gentle analeptic action helps counteract the apathy and inertia that can often delay convalescence—together with an excellent vitamin and mineral formula, in a satisfying 15% alcohol vehicle.

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And for your patient's sake, prescribe Alertonic in the convenient, economical one-pint bottle.

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Each 45 cc. (3 tablespoonfuls) contains: alcohol, 15%; pipradrol hydrochloride, 2 mg.; thiamine hydrochloride (vitamin B_1) (10 MDR*), 10 mg.; riboflavin (vitamin B_2) (4 MDR), 5 mg.; pyridoxine hydrochloride (vitamin B_6), 1 mg.; niacinamide (5 MDR), 50 mg.; choline,† 100 mg.; inositol,† 100 mg.; calcium glycerophosphate, 100 mg. (supplies 2% MDR for calcium and for phosphorus) and 1 mg. each of the following: cobalt (as chloride), manganese (as sulfate), magnesium (as acetate), zinc (as acetate), and molybdenum (as ammonium molybdate).

*Multiple of adult Minimum Daily Requirement supplied.
†The need for these substances in human nutrition has not been established.

Indications: 1. Functional fatigue such as that often associated with: a depressing life experience or stressful time of life; advancing years; convalescence; limited activity or confinement. 2. Poor appetite and vitamin-mineral deficiency as they occur in: patients having faulty eating habits; geriatric patients who are losing interest in food; patients convalescing from debilitating illness or surgery.

Dosage: Adults, 1 tablespoonful; children (over 15 years old), 1 to 2 teaspoonfuls; children (4 to 15 years old), 1 teaspoonful. To be taken three times daily 30 minutes before meals.

Contraindications: As with other drugs with CNS stimulating action, Alertonic is contraindicated in hyperactive, agitated or severely anxious patients and in chorea or obsessive compulsive states.

Side effects: Reports of overstimulation have been rare. Patients who are known to be unduly sensitive to the effects of stimulant drugs should be observed carefully in the initial stages of treatment.

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MOLECULAR REMODELING—

laboratory exercise or clinical necessity?

More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors —in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9,10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

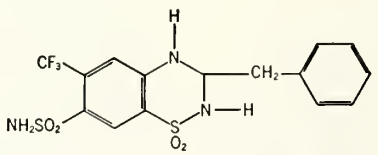
Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumethiazide a rather dramatic reduction in dose range is accomplished. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effective antihypertensive agent.



Contraindications: Severe renal impairment; previous hypersensitivity.

Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

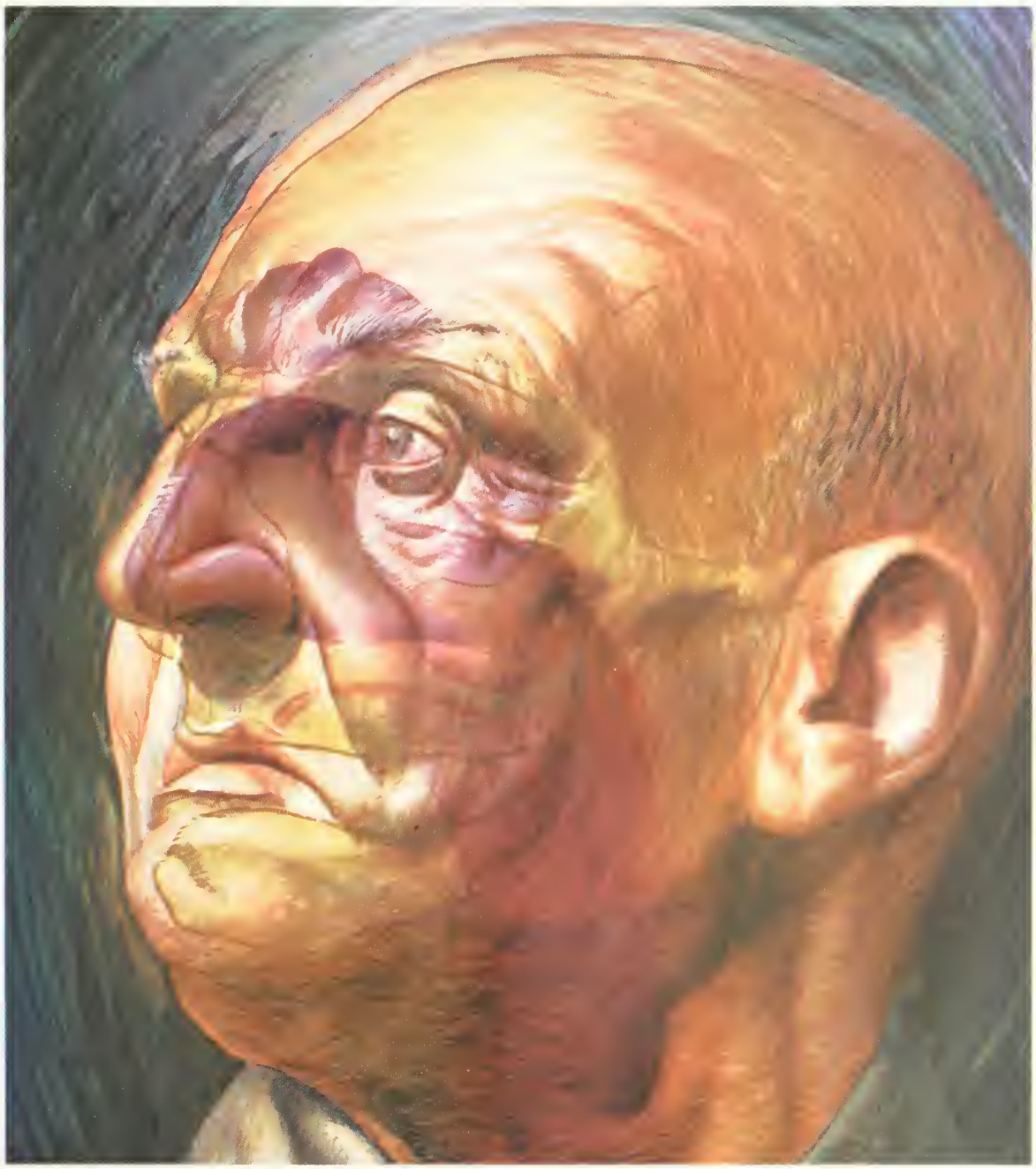
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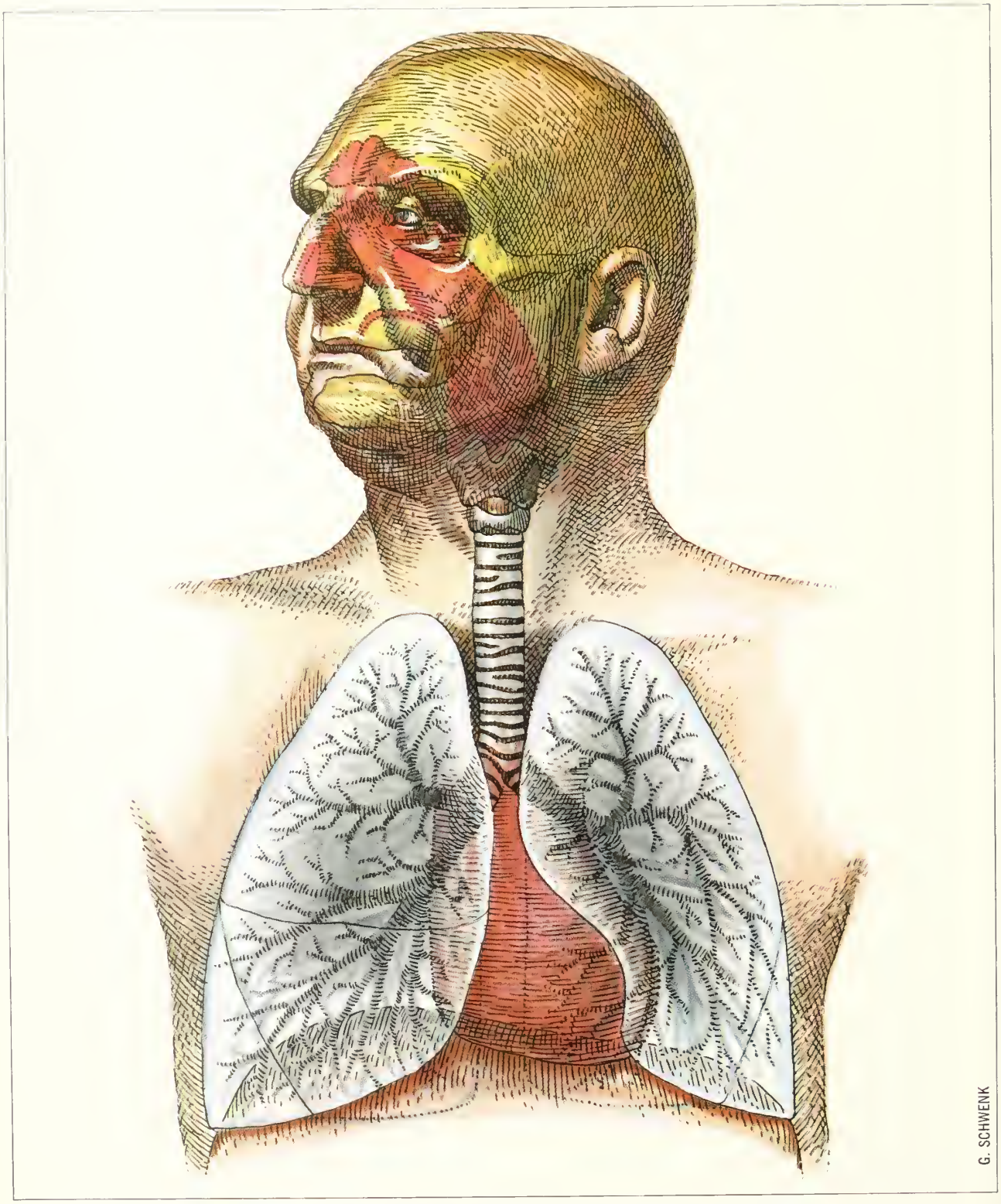
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this issue: the common cold and the aging patient

the common cold and the aging patient

Louis J. Vorhaus, II, M.D., F.A.C.P.



Effects of aging on the anatomic and physiologic aspects of the respiratory apparatus.

The chest becomes more fixed, less mobile and less elastic as the bronchial walls and thoracic ligaments lose elasticity. The diaphragm and intercostal muscles atrophy and weaken. The lungs become smaller, flabbier and weigh less, decreasing vital and total lung capacity, increasing residual volume and the alveolar dead space.

Sir William Osler described pneumonia as the welcome friend of the aged patient, because the patient with pneumonia usually died quietly. But today, the well-informed physician is an even better friend of the aging patient, since it is better to live than to die, no matter how quietly.

One of the first avenues of approach in the control of the hazards of respiratory disease in the aging patient is prompt and proper attention to the common cold or upper respiratory infection. The common cold may be the first step in the relatively short path to lower respiratory infection, broncho-pneumonia and death. This train of events occurs frequently among older persons. Indeed, pneumonia is one of the most common causes of their admission to hospitals and ranks high on the list of geriatric killers. Colds are more debilitating in elderly people and the aged are more likely candidates for secondary infections such as sinusitis and bronchitis. These infections, in turn, are more prone to lead to broncho-pneumonia, because of lowered resistance and anatomic and physiologic changes in the lungs of the elderly.

What is different about the respiratory tree of an aged person and that of an otherwise healthy younger adult? Aging certainly takes its toll on all parts of the body, affecting both anatomic and physiologic aspects of the respiratory apparatus. These changes are in part due to the wear and tear that occurs over the years; the repeated bouts of respiratory infection, long exposure to atmospheric pollutants, to occupational inhalants, smoking, malnutrition, obesity, inactivity and the development of other diseases which may affect the lungs.

With the passage of years, the lungs change. They become scarred and emphysematous and lose their compliance. The whole chest becomes more fixed, less mobile and less elastic.

The anatomic changes that occur in aging render the lungs less efficient. Tests of pulmonary function in senescence show a deterioration characterized by a decrease in vital capacity and total lung capacity, an increase in residual volume and alveolar dead space. Maximum breathing capacity is reduced and uniformity of ventilation deteriorates. These problems are often aggravated by the obstructed breathing, fever and secondary infection associated with the common cold, placing an additional stress on the entire cardiopulmonary reserve.

In addition, the efficiency of a cough is below par in older persons even though they are in good health. This is partly due to the decreased respiratory excursions and distensibility of the chest wall, and partly from loss of elasticity of bronchial walls which tend to make them collapse in a cough.

The elderly patient's resistance to infection is often reduced. Nutritional deficiencies are more common in aged people. There is some evidence to indicate that their capacity to respond to stress is less efficient. Finally, there is often relatively meager symptomatic response to acute disease. The absence of obvious or dramatic clinical signs and symptoms of severe illness is particularly dangerous because, coming as it

(concluded on following page)

it's a
comforting thing
to know



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Triaminic. Isn't that a comforting thing to know?

Each timed-release tablet contains:

Phenylpropanolamine hydrochloride	50 mg.
Pheniramine maleate	25 mg.
Pyrilamine maleate	25 mg.

Side effects: Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. Precautions: The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

(Advertisement)

does in a person whose defenses are weakened both locally and systemically, pulmonary disease may progress rapidly to irreversible stages before medical attention is sought. Respiratory infection is especially hazardous because the aged patient responds badly to hypoxia. Not only is his response to oxygen lack impaired, but the work of breathing, due to decreased compliance of the lung and increased stiffness of the thorax, is markedly augmented.
Many patients late in life are in a precarious and delicate cardiopulmonary balance which is easily decompensated from relatively minor insults such as colds and upper respiratory infections.

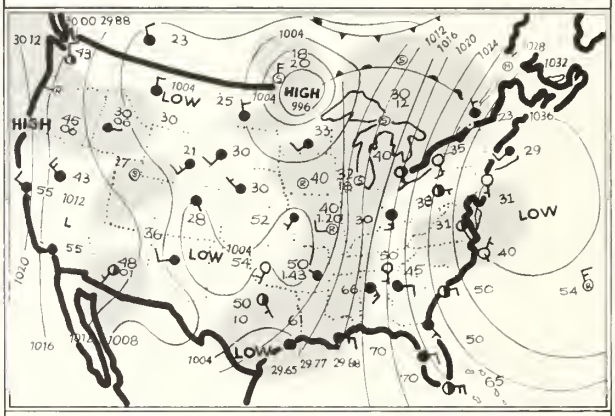
For all of these reasons, geriatricians long have stressed the importance of preventing respiratory insults. Today we have better ways of treating respiratory infection, improved techniques for clearing the lungs and bronchial tubes of secretions and better understanding of ways of improving ventilation. We possess a broader spectrum of antimicrobial agents including newer ones to deal with previously resistant organisms. Even so, the death rate from pneumonia is high in older people, and it is preferable to avoid the disease than to treat it. To do so, attention must be paid to the general maintenance of good health and all that implies, as well as to the prevention, elimination and treatment of associated conditions that predispose to or cause pneumonia such as chronic upper or lower respiratory infection, respiratory allergy, chronic sinusitis and exposure to inspired irritants.

Colds and other minor respiratory infections, which favor the development of broncho-pneumonia, should be treated vigorously and promptly, particularly those patients whose aging process has been accompanied by the development of chronic pulmonary disease. Upper respiratory passages should be cleared with decongestants. Sinuses should be drained adequately. And, when indicated, appropriate antimicrobial therapy should be instituted before serious infection of the lower respiratory tree supervenes.

In decades past it was understandable that physicians welcomed pneumonia for the aged patients because it offered them a quiet and peaceful demise. Today we recognize that in many cases, peaceful as it may have been, such deaths were often avoidable. With the current knowledge and understanding of the problems that respiratory infections impose on aging people, vigilant medical attention can often restore them to a vigorous, rewarding and productive life so that the many opportunities that exist today for people to enjoy their golden years are realized and not stolen by untimely death.

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Tussagesic® provides up to 24-hour coverage of the tough cold with a single timed-release tablet dosed morning, midafternoon and at bedtime. Coughs are broken up, runny and stuffed noses are cleared and pain is relieved.

Each Tussagesic® timed-release tablet contains:

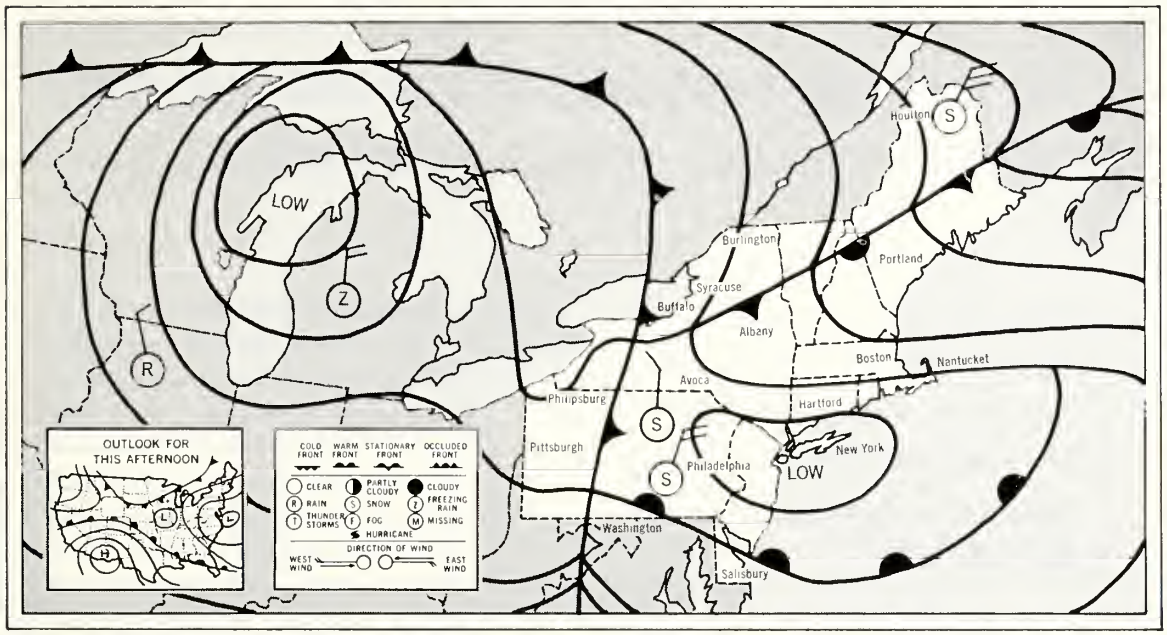
Triaminic® (phenylpropanolamine hydrochloride 25 mg.; pheniramine maleate 12.5 mg.; pyrilamine maleate 12.5 mg.)	50 mg.
Dextromethorphan hydrobromide	30 mg.
Terpin hydrate	180 mg.
Acetaminophen	325 mg.

Side effects: Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. Precautions: Patient should not drive a car or operate dangerous machinery if drowsiness occurs. Except under professional care, do not give to patients under 12 yrs. or those who have persistent cough, high fever, heart or thyroid disease, hypertension or diabetes or use for more than 10 days.

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REGIONAL WEATHER FORECAST

High Winds, Snow Storms and Much Colder Followed by Cough, Stuffed and Runny Noses and Aches and Pains.



Tussagesic breaks up coughs, quickly clears stuffed and runny noses and relieves aches and pains. Provide coverage of the tough cold for up to 24 hours with just a single timed-release tablet dosed morning, midafternoon and at bedtime.

each

Tussagesic®

timed-release tablet contains:

Triaminic® (phenylpropanolamine hydrochloride 25 mg., pheniramine maleate 12.5 mg., pyrilamine maleate 12.5 mg.)	50 mg.
Dextromethorphan hydrobromide	30 mg.
Terpin hydrate	180 mg.
Acetaminophen	325 mg.

Dosage: Adults—1 tablet, swallowed whole to preserve timed-release feature, in morning, midafternoon and at bedtime. *Side effects:* Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. *Precautions:* The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

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Norinyl® tablets

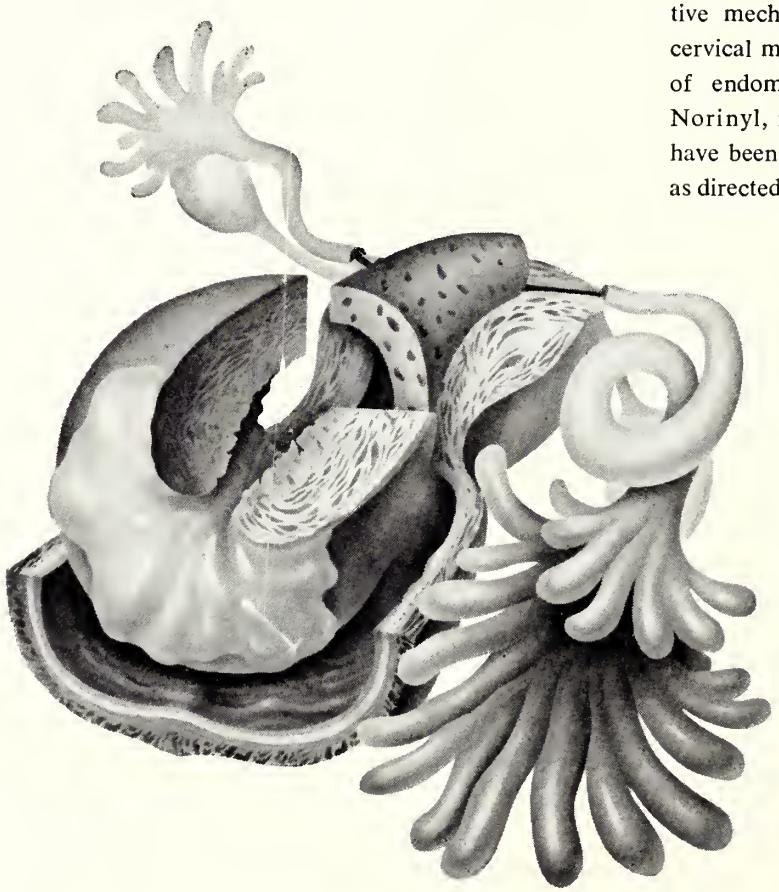
(norethindrone 2 mg ē mestranol 0 1 mg)

for multiple contraceptive action that has produced a record of unexcelled effectiveness

inhibition of ovulation by means of 2 time-proved hormonal agents

production of a cervical mucus hostile to sperm motility and vitality

creation of an endometrium unreceptive to egg implantation



no unplanned pregnancies

Norinyl provides multiple action for maximum assurance of success. It does not depend on ovulation inhibition alone for contraceptive effectiveness. The mechanism of action of combined hormonal therapy results in ovulation inhibition reinforced by other protective mechanisms, including a hostile cervical mucus[1-13] and an acceleration of endometrial changes.[1-3,7-16] With Norinyl, no unplanned pregnancies have been reported to date when used as directed.

plus important supportive benefits that help her through those critical early months of oral contraception

low incidence of side effects

Low incidence of BTB and spotting, nausea and amenorrhea tends to minimize side effect problems and increases patient cooperation.

no confusion about dosage

An unbreakable "confusionproof" package makes it easy to adhere to prescribed dosage schedule: individually sealed tablets numbered from 1 through 20 *plus* monthly calendar record enables patient to double-check dosage intake by day and corresponding tablet number.



Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.—mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

References: 1. Council on Drugs. JAMA 187:664 (Feb 29) 1964 2. Bryans, F. E.: Canad Med Ass J 92:287 (Feb. 6) 1965. 3. Goldzieher, J. W.: Med Clin N Amer 48:529 (Mar.) 1964 4. Cohen, M. R.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965. 5. Hammond, D. O.: Ibid. 6. Rice-Wray, E., Goldzieher, J. W., and Aranda-Rosell, A.: Fertil Steril 14:402 (Jul.-Aug.) 1963. 7. Goldzieher, J. W., Moses, L. E., and Ellis, L. T.: JAMA 180:359 (May 5) 1962. 8. Kempers, R. D.: GP 29:88 (Jan.) 1964. 9. Tyler, E. T.: JAMA 187:562 (Feb. 22) 1964 10. Rudel, H. W., Martinez-Manautou, J., and Maqueo-Topete, M.: Fertil Steril 16:158 (Mar.-Apr.) 1965. 11. Flowers, C. E., Jr.: N Carolina Med J 25:139 (Apr.) 1964. 12. Goldzieher, J. W.: Appl Ther 6:503 (June) 1964. 13. The Control of Fertility. Report adopted by the Committee on Human Reproduction of the American Medical Association. JAMA 194:462 (Oct. 25) 1965. 14. Flowers, C. E., Jr.: JAMA 188:1115 (June 29) 1964. 15. Merritt, R. I.: Appl Ther 6:427 (May) 1964 16. Newland, D. O.: Paper presented at Symposium on Low-Dosage Oral Contraception, Palo Alto, Calif., July 15, 1965. Reported in Med Sci 16:26 (Nov.) 1965.

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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

Contraindications: Patients hypersensitive to antihistamines. Not recommended for use during pregnancy.

Precautions: Until the patient's response has been determined, he should be cautioned against engaging in operations requiring alertness. Administer with care to patients with cardiac or peripheral vascular diseases or hypertension.

Side Effects: Hypersensitivity reactions including skin rashes, urticaria, hypotension and thrombocytopenia have been reported on rare occasions. Drowsiness, lassitude, nausea, giddiness, dryness of the mouth, mydriasis, increased irritability, or excitement may be encountered.

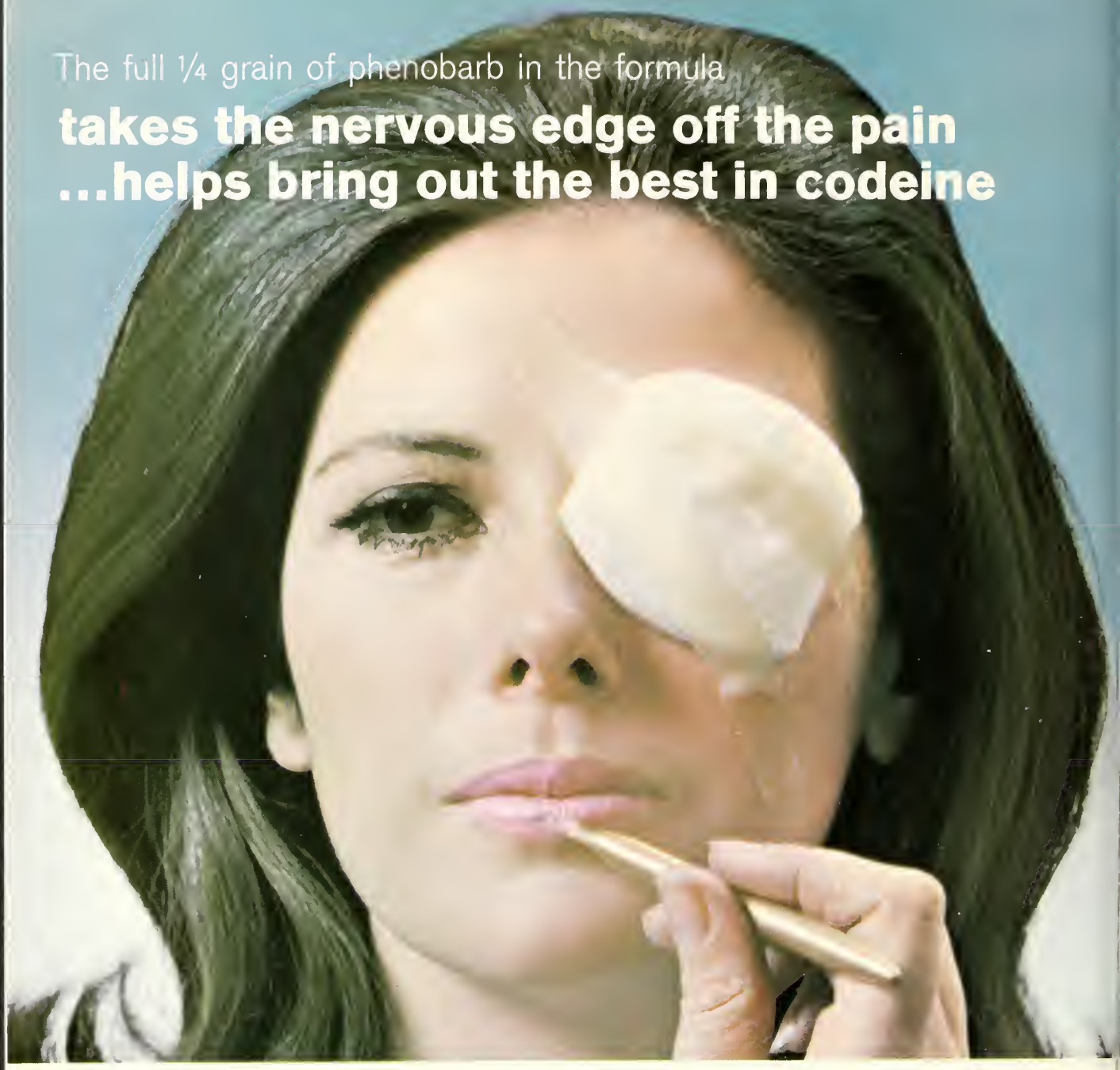
Dosage: 1 Extentab morning and evening, or as needed.

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Also available: Dimetapp® Elixir for conventional *t.i.d.* or *q.i.d.* dosage. See package insert for further details.

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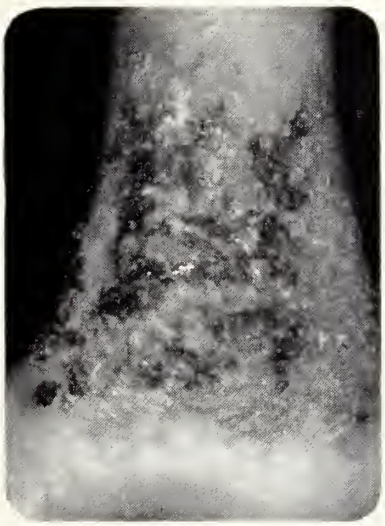
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Side Effects: Side effects are uncommon—nausea, constipation, and drowsiness have been reported.

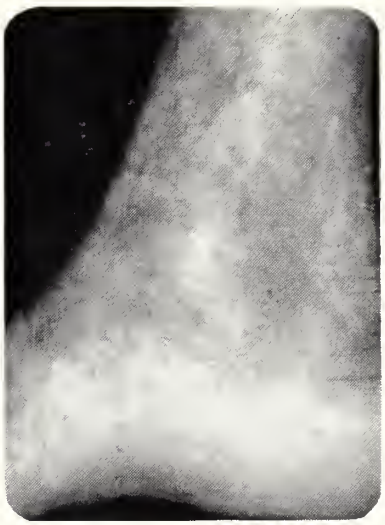
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After treatment—with ARISTOCORT Topical Ointment 0.1% for two weeks

ARISTOCORT® Triamcinolone Acetonide Topicals have proved exceptionally effective in the control of various forms of childhood eczema: allergic, atopic, nummular, psoriatic, and mycotic.

In most cases responsive to topical ARISTOCORT, the 0.1% concentration is sufficiently potent. The 0.5% concentration provides enhanced topical activity for patients requiring additional potency for proper relief.

Administration and Dosage: Apply sparingly to the affected area 3 or 4 times daily. Some cases of psoriasis may be more effectively treated if the 0.1% Cream or Ointment is applied under an occlusive dressing.

Contraindications: Tuberculosis of the skin, herpes simplex, chicken pox and vaccinia.

Precautions and Side Effects: Do not use in the eyes or in the ear (if drum is perforated). A few individuals react unfavorably under certain conditions. If side effects are encountered, the drug should be discontinued and appropriate measures taken. Use on infected areas should be attended with caution and observation, bearing in mind the potential spreading of infection and the advisability of discontinuing therapy and/or initiating antibacterial measures. Generalized dermatological conditions may require systemic corticosteroid therapy. Steroid therapy, although responsible for remissions of dermatoses, especially of allergic origin cannot be expected to prevent recurrence. The use over extensive body areas, with or without occlusive nonpermeable dressings, may result in systemic absorption. Appropriate precautions should be taken. When occlusive nonpermeable dressings are used, miliaria, folliculitis and pyodermas will sometimes develop. Localized atrophy and striae have been reported with the use of steroids by the occlusive technique. When occlusive nonpermeable dressings are used, the physician should be aware of the hazards of suffocation and flammability. The safety of use on pregnant patients has not been firmly established. Thus, do not use in large amounts or for long periods of time on pregnant patients.

Packages: Tubes of 5 Gm. and 15 Gm.; ½ lb. jar.

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against the usual gram-negative urinary pathogens

Why use five...where **one** will do?



In a recent 217-patient hospital study,[1] urinary tract infections were treated with a variety of widely prescribed antimicrobial agents including: a sulfonamide (40 patients), chloramphenicol (20 patients), nitrofurantoin (33 patients), nalidixic acid (30 patients), tetracycline (27 patients), colistimethate sodium (22 patients)... and 2 combinations of 5 agents each (45 patients). The 2 combinations were selected to afford maximal theoretical antibacterial coverage against the usual urinary pathogens. They were (1) tetracycline, chloramphenicol, nitrofurantoin, ristocetin and polymyxin B; and (2) tetracycline, chloramphenicol, erythromycin, nitrofurantoin and colistimethate sodium.

This clinical study shows that the two combinations of antibiotics were not superior to some of their single components. The authors point out that antibiotic antagonism often negates theoretical advantages of multiple therapy. Coly-Mycin Injectable (colistimethate sodium) was one of the single components that was shown to be equal to the combinations and eradicated bacteriuria in two-thirds of the patients.

Theoretical choice of multiple antibacterial therapy has been shown to be no more effective than one well-chosen agent which also offers least patient exposure to possible side reactions, toxicities, allergic manifestations and higher drug costs.

1. McCabe, W. R., and Jackson, G. G.: New England J. Med. 272:1037, 1965.

in gram-negative urinary tract infections often the single well-chosen agent



Coly-Mycin® Injectable
(colistimethate sodium)

Indications: Especially indicated for the treatment of severe acute and resistant chronic urinary tract infections due to sensitive strains of gram-negative organisms. Also indicated in respiratory tract, surgical, wound and burn infections and in septicemia due to sensitive organisms. Particularly indicated when any of these infections are caused by sensitive strains of *Pseudomonas aeruginosa*.

Adverse Reactions: Occasional reactions such as circumoral paresthesias, tingling of the extremities, pruritus, vertigo or dizziness may occur. Reduction of dosage may alleviate symptoms. Therapy need not be discontinued, but such patients should be observed with extra care.

Warning: Patients should be cautioned not to drive vehicles or use hazardous machinery while on therapy.

Precautions: In cases of impaired or suspected renal impairment, use with greater caution and reduce dosage in proportion to extent of impairment. Transient elevations of BUN have been reported. As a routine precaution, appropriate blood studies should, therefore, be made during prolonged therapy.

As with all polypeptides, the possibility of muscular weakness, including apnea, due to inadvertent overdosage or normal dosage in the presence of impaired renal function, should not be overlooked. In cases of apnea, medication should be promptly discontinued and assisted respiration given until serum levels fall and normal breathing is restored.

Other antibiotics, such as kanamycin, streptomycin, dihydrostreptomycin, polymyxin, and neomycin, may also have varying neurotoxic or nephrotoxic potential. They should be used with great caution concomitantly with Coly-Mycin Injectable (colistimethate sodium).

For deep intramuscular injection only.

Dosage: By the I.M. route only, in 2 to 4 divided doses ranging from 1.5 to 5 mg./Kg./day (0.7 mg. to 2.3 mg./lb./day). Average adult dose is 2.5 mg./Kg./day (1.1 mg./lb./day). In the presence of bacteremia, septicemia, or other serious infection, greater than average doses may be required; however, maximum daily doses should not exceed 5 mg./Kg. (2.3 mg./lb.) where renal function is normal.

Not recommended against Proteus.

Colistin is also available (as colistin sulfate) in: Coly-Mycin® Pediatric for Oral Suspension (not for systemic use), and Coly-Mycin® Otic with Neomycin and Hydrocortisone.

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Warn against hazardous occupations requiring complete mental alertness. Use caution in administering to addiction-prone patients or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In elderly and debilitated and in children over five, limit dosage to smallest effective amount, increasing gradually as needed and tolerated. In general, concomitant use with other psychotropics is not recommended. Paradoxical reactions have been reported in psychiatric patients and hyperactive aggressive children. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Observe usual precautions in presence of impaired renal or hepatic function, impending depression and suicidal tendencies.

Adverse reactions: Drowsiness, ataxia and confusion may occur, especially in elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. Syncope occurs rarely. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis, jaundice and hepatic dysfunction) may develop occasionally, making periodic blood counts and liver-function tests advisable during protracted therapy. Individual maintenance dosages should be determined.

Dosage: *Oral*—Adults: Mild to moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d.; severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d.

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THE JOURNAL of The Maine Medical Association

VOLUME 57 DECEMBER, 1966 NUMBER 12

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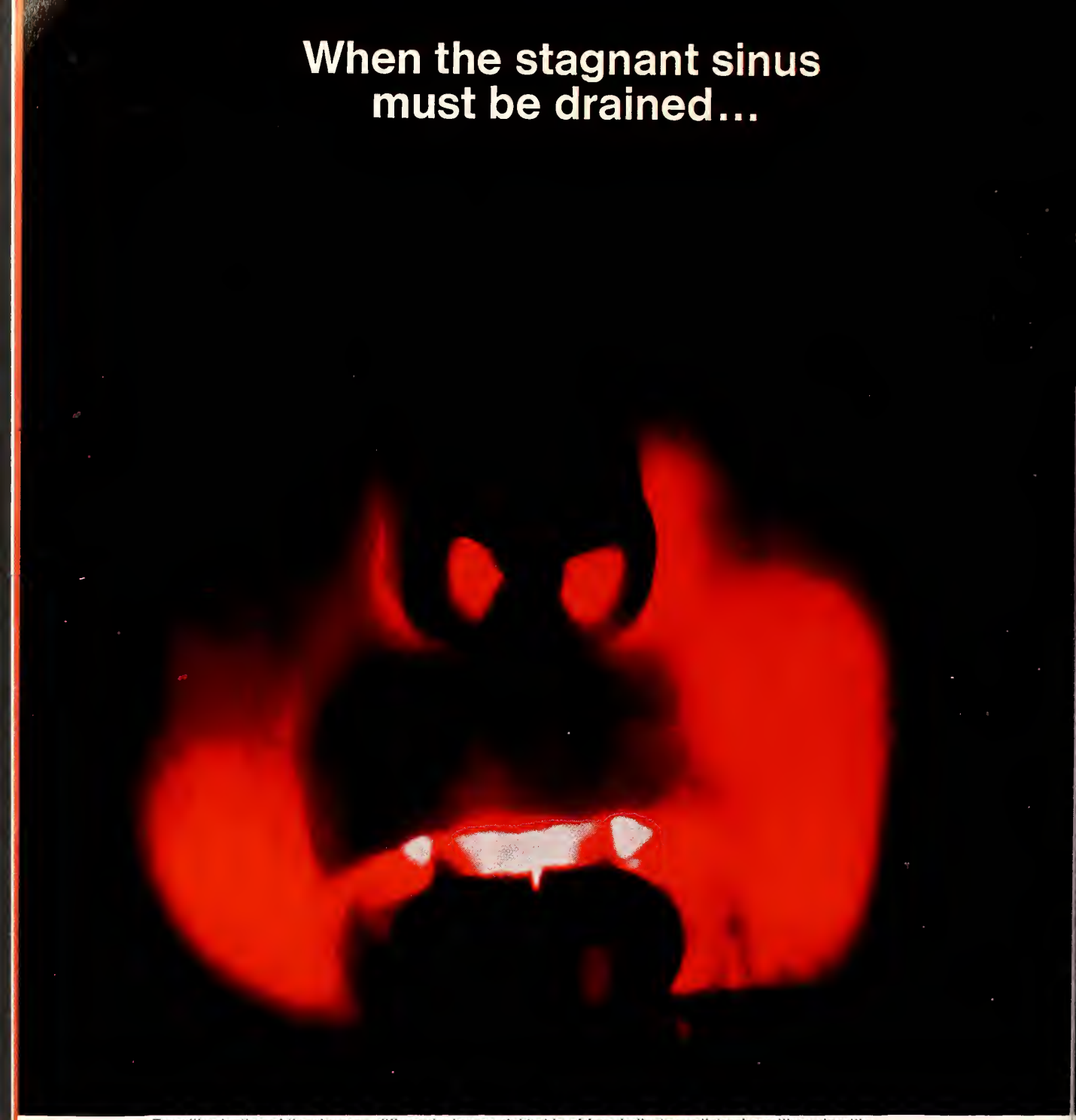
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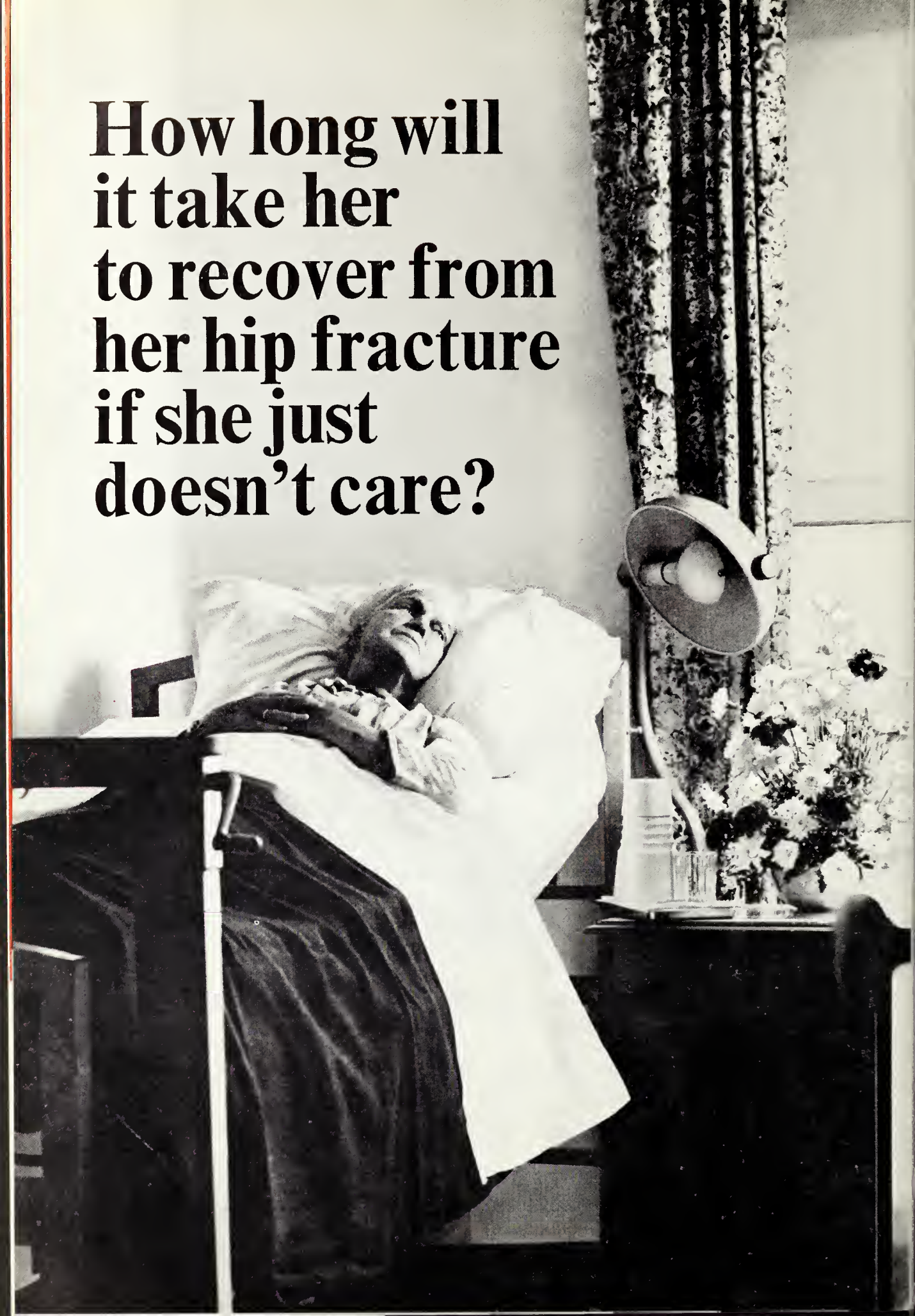
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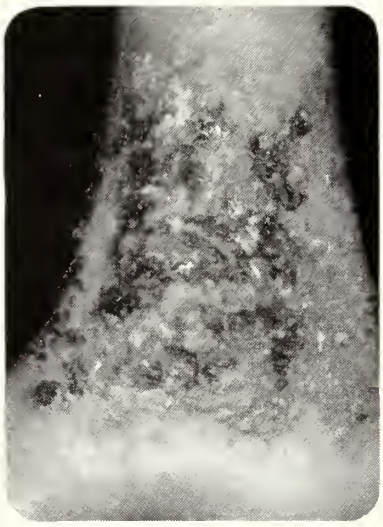
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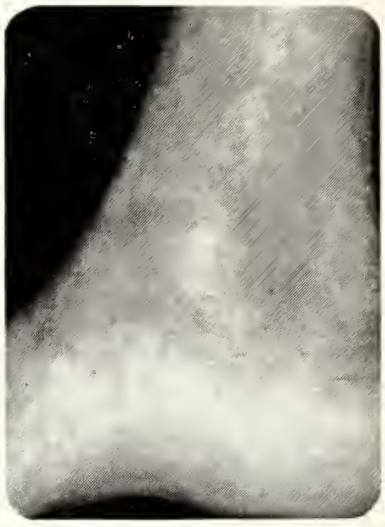
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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

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Side Effects: Hypersensitivity reactions including skin rashes, urticaria, hypotension and thrombocytopenia have been reported on rare occasions. Drowsiness, lassitude, nausea, giddiness, dryness of the mouth, mydriasis, increased irritability, or excitement may be encountered.

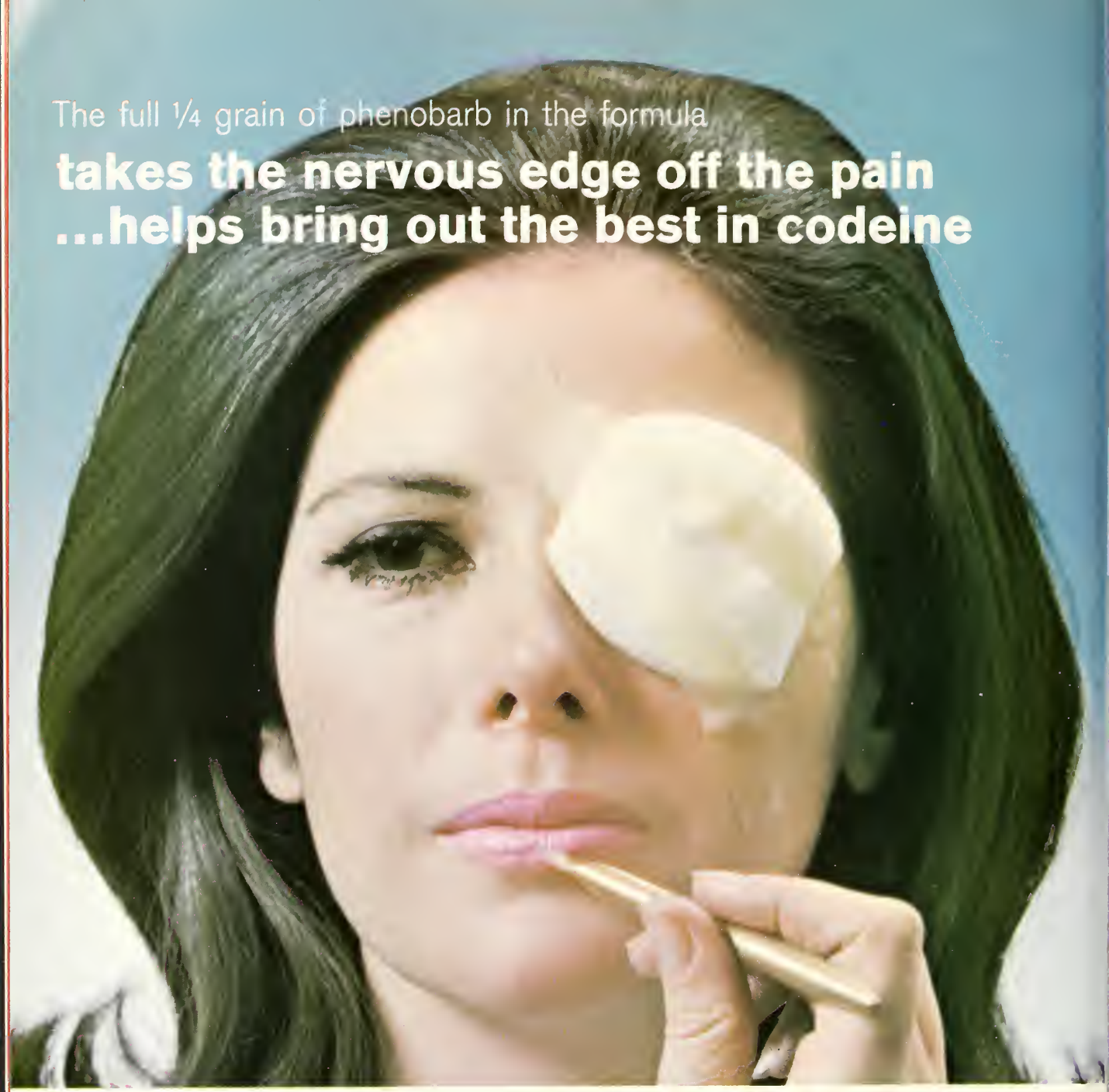
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SQUIBB NOTES ON THERAPY



Breast-feeding and the "modern mother"

Despite a mild resurgence of interest in the importance of breast-feeding a few years ago, many women today do not choose to nurse their young. This is for a variety of reasons—social, economic, cultural and sometimes medical. In such cases the physician's task is to find the most suitable means of preventing lactation and easing the pain of breast engorgement.

The means of therapy

The value of hormone therapy for this indication is of course well established. Both androgen and estrogen are known to inhibit the production and secretion of the lactogenic hormone by the anterior pituitary. As estrogen levels decline sharply at parturition, lactogenesis is established. When androgen and estrogen are administered to the patient before the release of the lactogenic hormone lactation and breast engorgement are usually prevented.

The time of therapy

The time of administration of this combined medication is crucial; it must be given early enough to suppress the pituitary prolactin and last long enough to permit physiologic readjustment during the puerperium. Excellent results are most often seen when therapy is administered before the onset of the second stage of labor.

However, factors other than effectiveness must also be considered. The agent selected should not interfere in any way with parturition, subsequent uterine involution and the restoration of normal ovarian cyclic function. Furthermore, it should not cause rebound breast engorgement or other manifestations of hormonal imbalance.

A balanced formulation

Providing single-dose therapy for the prevention of lactation and breast engorgement, Deladumone OB is a potent androgen-estrogen combination with a prolonged action. The optimal balance of androgenic and estrogenic hormones achieved in this preparation minimizes the disadvantages inherent in single hormone therapy, such as rebound breast engorgement. Involution of the uterus and resumption of menstrual cycles are not affected.

As reported in a recent published study (Roser, D. M.: Obstet. & Gynec. *27*:73, 1966), Deladumone OB provided good suppression of breast engorgement in 95.3% and suppression of lactation in 81.1% of 86 obstetrical patients. These results are in general agreement with those of many earlier investigations; in several studies this injectable androgen-estrogen combination proved to be superior to oral medication.

Dosage:

As a single injection of 2 cc. before the onset of the second stage of labor.

Contraindications:

Established or suspected mammary cancer or genital malignancy.

Precautions and Side Effects:

Certain patients may be unusually responsive to either estrogenic or androgenic therapy. In such individuals virilization, uterine bleeding or mastodynia may occur.

Supply:

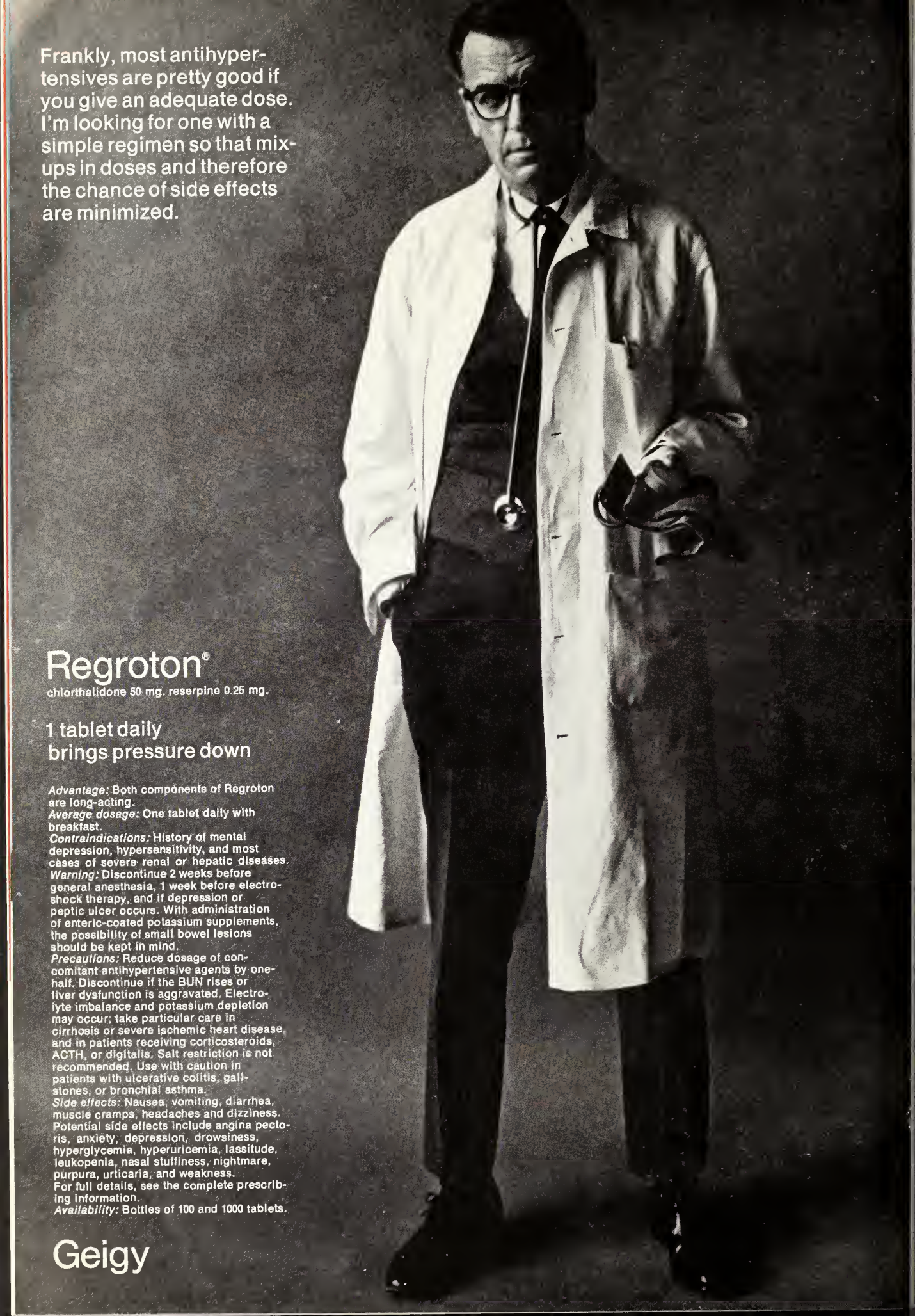
Deladumone OB, providing 180 mg. testosterone enanthate and 8 mg. estradiol valerate per cc., is available in 2 cc. Unimatic® disposable syringes and in 2 cc. vials. Both preparations are dissolved in sesame oil, with 2% benzyl alcohol as a preservative. *Before use, consult product literature for full prescribing information.*

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Precautions: Reduce dosage of concomitant antihypertensive agents by one-half. Discontinue if the BUN rises or liver dysfunction is aggravated. Electrolyte imbalance and potassium depletion may occur; take particular care in cirrhosis or severe ischemic heart disease, and in patients receiving corticosteroids, ACTH, or digitalis. Salt restriction is not recommended. Use with caution in patients with ulcerative colitis, gallstones, or bronchial asthma.
Side effects: Nausea, vomiting, diarrhea, muscle cramps, headaches and dizziness. Potential side effects include angina pectoris, anxiety, depression, drowsiness, hyperglycemia, hyperuricemia, lassitude, leukopenia, nasal stuffiness, nightmare, purpura, urticaria, and weakness.
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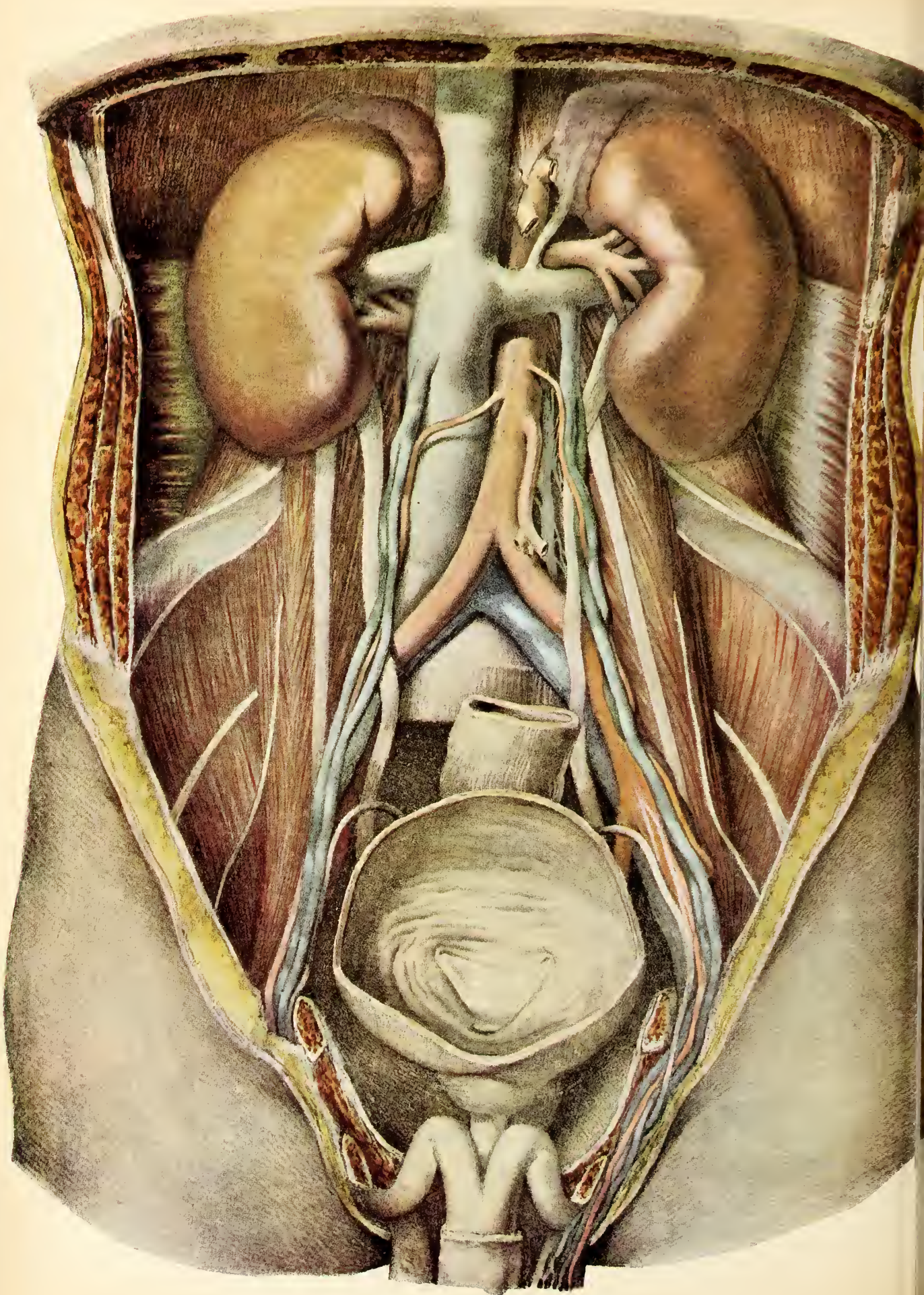
Use cautiously in individuals with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Caution patients who operate machinery or motor vehicles that drowsiness may result.

Each Novahistine LP tablet contains: phenylephrine hydrochloride, 25 mg., and chlorpheniramine maleate, 4 mg.

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Normally produces high and persistent levels in blood and high concentrations in bile and urine.

Significant inherent stability.

**Exclusive of penicillinase-producing bacteria.*

Indications: Urinary tract infections, especially those caused by *E. coli*, *Proteus mirabilis*, and *Streptococcus faecalis* and *viridans;* respiratory infections caused by *H. influenzae*, pneumococci, streptococci, and nonpenicillinase-producing staphylococci; and gastrointestinal infections caused by *Shigella* and *Salmonella*, including *Sal. typhosa.*



Contraindications: Hypersensitivity to penicillin; infections due to penicillinase-producing staphylococci and other penicillinase-producing bacteria.

Precautions: If allergic reaction occurs, discontinue ampicillin and administer epinephrine, corticosteroids, antihistamines and/or pressor amines as indicated. Transient moderate elevation of SGOT values of undetermined significance was noted in a few infants. Liver and kidney function as well as hematopoietic tests are advisable during therapy, particularly in infants. As with other antibiotics, precautions should be taken against gastrointestinal superinfection. Safety for use in pregnancy has not been established.

Adverse Reactions: Occasional mild side effects as urticaria, skin rash, pruritus, diarrhea, nausea and vomiting. There have been no reports of blood dyscrasias, liver or kidney damage. Anaphylaxis has been reported.

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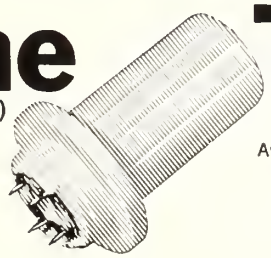
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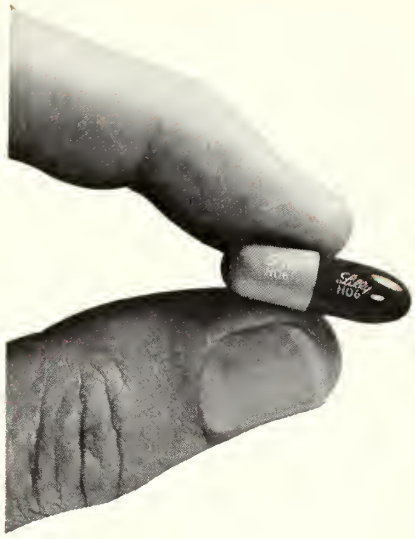
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ling or measurements of ventilation so indicate, ventilation should be supported either by bag and mask or suitable mechanical ventilator.

We are now using the third generation of analeptics of which doxapram is perhaps the best known. These drugs act quite specifically on the reticular formation and are particularly useful in differentiating respiratory center depression from neuromuscular blockade in the hypoventilating postoperative patient. These drugs should be used in intermittent single dose and not by continuous I.V. drip and must be used with great caution in the patient with pulmonary disease or incipient congestive heart failure.

Allergic Emergencies

Treatment of hypoxia in allergic emergencies also follows the rule of the ABC of resuscitation. Attention to airway, breathing and circulation, accompanied by the judicious use of epinephrine will suffice in most emergencies. In status asthmaticus more vigorous resuscitation is required.

1. 100% O_2.
2. Increase alveolar ventilation by manual methods or the use of a volume controlled respirator.
3. Treat acidosis with 100 – 400 meq. of $NaHCO_3$ in 500cc 5% D/W according to arterial ph.
4. Aminophylline 500 – 1000 mgm I.V.
   Prednisolone 60 – 80 mgm.
   Occasionally epinephrine – 1 mgm in 250 – 500cc 5% D/W.
   Deep ether or halothane have been advocated but are extremely dangerous in severely hypoxic patients.

Myocardial Infarction

The treatment regime of hypoxia due to myocardial infarction relates to the presence or absence of shock. There is some apparent benefit in use of O_2 in uncomplicated myocardial infarction. In patients with cardiogenic shock there is urgent need for O_2 in very high concentration. In these patients about 25% of the output of the right heart passes through vessels inaccessible to pulmonary gas exchange. The cause for his shunting is unknown but may be due to collapse, blockage or edema flooding of alveoli.

The plastic face mask with flutter valves will produce an inspired concentration of O_2 in the range of 60 – 80% at a flow of 8 – 10 L/min. The O_2 tent will provide a concentration of 45 – 50% if canopy is not opened. Though there are many drawbacks to the use of the tent it is for psychological reasons often better tolerated by the patient.

Cardiac Arrest. If hypoxia is due to cardiac arrest the following regime is recommended by the American Heart Association:

1. Airway — Open by tilting head back.
2. Breathe — Mouth to mouth approximately 12 times per minute.
3. Circulate — Depress sternum once per second.
4. Drugs — Administer
   Epinephrine – 0.5 – 1.0 mgm. I.V. or I.C. Sodium bicarbonate – 3.75 gm./50cc I.V.
   Repeat every 10 minutes until pulse returns.
5. EKG — Determine if ventricular fibrillation or asystole.
6. Fibrillation treatment:
   External defibrillation
   AC – 440-880V
   DC – 100-400 Watt/sec

Restoration of pulse should be followed by hypothermia, intensive care and prolonged controlled ventilation as indicated.

205 French Street, Bangor, Maine



The Management of Shock

EDWARD B. BABCOCK, M.D.*

A definition of shock is impaired effective tissue perfusion of blood. Perfusion is what is treated, not primarily the blood pressure. The signs of shock may be variable and inconstant. For instance, the pulse may be slow even in profound shock; the skin may be warm and dry. Impaired perfusion is where the damage lies. Coronary and brain circulation are the last to go. Renal circulation with early vasoconstriction is one of the first. Centrilobular hepatic necrosis occurs early.

TABLE 1

SHOCK: INADEQUATE EFFECTIVE TISSUE PERFUSION OF BLOOD
Hypotension
Increased pulse
Tachypnea
Apprehension, confusion
Pallor, cyanosis, coldness, blanching
Impaired perfusion of vital organs
Kidney
Liver
Intestine
Coronary circulation
Brain

Though some knowledge of the cause of shock is needed to plan management and should be searched for, and any bleeding promptly controlled, the following outline is suggested for the management of persistent shock and emphasizes three major components:

Respiratory support is the first consideration in treating shock. A clear airway is established either by positioning, oral airway, endotracheal tube or tracheostomy with suctioning as needed. Breathing assistance with an Ambu-type bag or mechanical respirator may improve ventilation. Increased oxygen concentration in the inspired air is almost always desirable.

Secondly, evaluation of central hemodynamics in persistent hypotension is fundamental to management. In a physiologic sense shock is related to one or another of HYPOVOLEMIA-CARDIAC INSUFFICIENCY-DEFECTIVE VASCULAR TONE. Considering a given vascular space –

a. (Hypovolemia) If fluid content is lost, eventually a point will be reached where even increased pumping action cannot maintain effective perfusion. Examples of this hypovolemic shock are hemorrhage, burns, trauma, and dehydration.

b. (Cardiac insufficiency) Inadequacy of the cardiac pumping action even with a normal space and volume may impair perfusion. In this group are left ventricular failure or myocardial infarction, cardiac arrhythmias, and obstruction to venous return such as cardiac tamponade or tension pneumothorax.

c. (Defective vascular tone) If the intravascular space is enlarged by defective vascular tone even with normal volume and pump action, inadequate perfusion again may result. This occurs with severe neurologic injuries, drug intoxication such as barbituates or narcotics, severe hypoxia, hepatic cirrhosis, and some cases of endotoxin shock and sepsis.

A measure of central events may help to define which is present. Perhaps the area of most agreement in shock is the usefulness of central venous pressure. This is an easy procedure and available to a hospital of any size.

The needle is placed in an antecubital vein (or external jugular or subclavian vein) and the catheter, still in a sterile transparent plastic envelope, is threaded through the needle to the superior vena cava. The needle is then withdrawn around the catheter and attaches to the adapter at the end. The plastic envelope is removed. A long Y tube attaches to the adapter. One arm of the Y extends from a bottle of IV solution. The other arm extends along the manometer scale for reading the central venous pressure. The zero point of the scale is placed at the midaxillary line at the 2nd interspace with the patient flat. A bottle of fluid is used to fill the system and maintain a drip between measurements. The great value of C.V.P. is the continuous information provided, regardless of cause, on how the heart is handling a particular or changing vascular volume. It guides the rate of administration and total volume that can be given without overloading. Examples of its usefulness might be:

a. If C.V.P. is over 10 centimeters of water initially, the heart is already unable to handle the vascular volume and venous return. No more fluid should be given until cardiac efficiency is improved to reduce C.V.P. even though hypovolemia may also be present. Digitalis, correction of arrhythmias and under certain circumstances cardiac stimulants might be used.

b. If C.V.P. is below 10 centimeters, especially if below 5 centimeters, the heart is handling its volume load effectively and fluids may be safely given, particularly since C.V.P. monitoring will detect early any fluid overloading.

C.V.P. also provides direct access to the active circulation for rapid fluid administration or prompt withdrawal of overload. By comparison, peripheral veins, because of size and intervening valves, do not accurately reflect central pressure or rapid changes especially in shock.

Thirdly is urinary output. The value of a measured

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output justifies catheterization particularly since obstruction with proximal infection and bacteremia is commonly the cause of shock. With outflow tract established, output will then measure urine formation Generally speaking a good urine output in the absence of diuretic therapy indicates that circulation is adequate. To regain adequate urine formation decreased intravascular volume and electrolyte imbalance need, if present, to be replenished first. Then if urine output remains less than 10 milliliters per hour an osmotic diuretic such as Mannitol may be given. Five percent Mannitol infusion is continued until either adequate urine volume (preferably in excess of 40 milliliters per hour) occurs or there is rising central venous pressure without increased urine flow. If IV fluids need be restricted, a 22% solution may be given, but the amount has to be limited since the hypertonic solution may draw more fluid into the vascular space. Mannitol may be of particular value in transfusion reactions and to prevent anuria in crush injuries where the plasma contains large amounts of circulating hemoglobin and myoglobin pigment which precipitate out in the renal tubules. If urinary output is then not improved and the condition of shock is reasonably correctable, peritoneal dialysis may tide the patient over until renal function returns.

There is evidence that capillary circulation is slowed in shock and that this may be related to increased viscosity of blood, RBC sludging and microcoagulation in small post capillary veins. Generally speaking in hypovolemic shock all volume expanders including whole blood increase cardiac output at least transiently, but since whole blood further increases viscosity, as well as risks hepatitis and transfusion reactions, it is not chosen unless there has been serious hemorrhage. Plasma and normal human serum albumin are both effective volume expanders. Low viscosity Dextran (average M.W. about 40,000), soon to be available, may be the solution of choice in most situations. By lowering plasma viscosity it may improve flow properties and with this molecular weight of 40,000 may have a duration of four or more hours in shock. The amount of low viscosity dextran is at present limited to 1500 cc as a 10% solution in saline or 5% D/W. In burn cases particularly to prevent shock, there is evidence for the continuous infusion of large amounts of Ringer's solution with lactate "fast enough to keep the patient safely and comfortably alive, making urine, and oriented, yet not so much to cause a significant rise in C.V.P." This and saline solution may also be used as emergency expanders but are lost rapidly from the circulation. Commercial high viscosity Dextran (M.W. 80,000) is an excellent expander but with its high molecular weight may further impair capillary flow. This is the 6% Dextran solution in most hospitals now.

The usefulness or lack of same of vasopressor and vasodilator drugs is not yet settled. If used, a drug should be matched to the individual problem, realizing that severe shock will not be corrected by one of these drugs alone.

TABLE 2

FLUIDS FOR VOLUME REPLACEMENT

- Low Viscosity Dextran
- Plasma
- Whole Blood
- Commercial Dextran
- IV Solutions
  - Ringer's with Lactate
  - Saline

TABLE 3

	Vasoconstrictor	*Cardiac Stimulant*	*Vasodilator*
Epinephrine		+++	+
Isuprel®		+++	+++
Levophed®	+++	+	
Aramine®	+++	+	
Neo-Synephrine®	++		
Vasoxyl®	++		
Angiotensin	++++		

It is generally agreed that in severe shock there is usually vasoconstriction that is often intense. Vasoconstrictor drugs perpetuate this already excessive effect. This is particularly true in hypovolemic or cardiogenic shock, and often endotoxin shock. Isuprel, being a vasodilator and a potent cardiac stimulant, has received considerable favorable study but is not without side effects. Shock with defective vascular tone may need a vasoconstrictor, particularly if the drug also has a cardiac stimulant effect (Aramine, Levophed), but keep in mind that vasodilation in the skin may be accompanied by vasoconstriction in other more vital areas. There being no present method for accurately measuring actual tissue perfusion the questions remain unsettled whether (1) a vasoconstrictor which is also a cardiac stimulant, such as Aramine or Levophed, might actually increase net flow despite the increased vasoconstriction, or (2) whether it is safe to risk further fall in blood pressure by dilating vessels to improve flow. For the most part vasoconstrictors are used as a last resort.

The value of adrenal steroids in shock remains unproved. There is reported to be improved cardiac efficiency and vascular tone with a potentiating effect to vasopressor drugs. Although not everybody agrees with their value, steroids are generally considered part of the treatment of bacterial endotoxin shock and overwhelming sepsis. If they are used, amounts in excess of 1000 milligrams per 24 hours are given. Shock associated with acute adrenal insufficiency remains the most specific indication for adrenal steroids.

Antibiotics, without waiting for culture reports, are started in shock related to sepsis, the choice being assisted by gram stain or the likelihood of organisms depending on the location of the infection. With a urinary tract infection, particularly with obstruction or a recent GU procedure, gram negative bacteremia is common.

Selection of an initial antibiotic in this situation varies in different clinics from Tetracycline alone to combinations of Tetracycline, Streptomycin and Chloromycetin.® With gram positive smears resembling staphylococci it is safer to start a penicillinase resistant drug like Oxacillin or Methicillin.

In untreated shock, metabolic acidosis develops rapidly. Tissue hypoxia increases anaerobic pathways of glucose metabolism, and thus excess lactate formation and hydrogen ion retention, with resultant lactic acidosis. The pH may be improved by sodium bicarbonate intravenously. But the correction of excess lactate formation depends primarily on the restoration of normal tissue perfusion and oxygenation.

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The Unconscious Patient

Diagnosis and Emergency Treatment

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When first approaching the bedside of an unconscious patient, my ears and eyes are tuned for specific facts from family, physician, nurses, technicians and charts. The following questions are pursued:

Is there any evidence for head trauma?
Has the patient had seizures?
Is the patient diabetic? Does he take insulin–when did he last have some?
What drugs has he had?
Does he have any of the things to suggest chronic lung disease?
Is he an alcoholic, is there evidence of liver disease?
Has he ever had any operations for endocrine disorders?
What laboratory and x-ray data are already known?

Being tuned in for any of these facts, I head straight for the bedside and check the vital signs and listen for the movement of air in and out of the chest. If the blood pressure or respiratory apparatus need help, this is provided immediately in the ways described by previous speakers. Having done that, I do a good physical examination including neurological, not letting the need for speed result in the haste that omits those scars or lacerations buried in the hair, the changing pupils and eyegrounds, the occasional spider, etc. After doing the physical and establishing at least temporary support of the cardio-respiratory systems, I take a few moments to pursue the questions mentioned that were in my mind as I moved to the bedside.

Then I think anatomically, moving from head to toe collecting crucial data to alert me as I proceed to get it:

Region	*Disease Suspected*	*Crucial Data*
Head	– Meningitis, abscess, hematoma or hemorrhage, hypopituitarism.	Neurological exam, skull films, L.P., temperature, skin.
Neck	– Myxedema, carotid insufficiency, airway obstruction.	Temperature, pCO_2, carotid pulses, bruit, inspection of airway.
Lungs	– Respiratory failure – associated with obstructive lung disease, drugs, pneumonia, lung abscess, pneumothorax.	Chest examination, pCO_2, pO_2, pH, minute volume, x-ray.
Heart	– Hypotension, arrhythmia.	Blood pressure, pulse, rhythm.
Abdomen	– Pancreatitis, perforated viscus, ruptured spleen, liver failure.	Amylase or diastase, abd. x-ray, abd. paracentesis, bilirubin, LDH, SGPT.
Kidneys	– Acute renal failure, or chronic, superimposed on other systemic dis.	Serum pH, BUN, Urine output.
Blood	– Ketoacidosis, hypoglycemia. Salicylate intoxication.	Serum ketones, pH, blood &/or urine sugar. Serum salicylates, pH, urine phenestix.

Barbiturate intoxication.	Serum level, pCO2.
Ethanol intoxication.	Examination, history, serum level.
Carbon monoxide intoxication.	Examination, oxygen content (not tension). History of exposure.
Clotting defect.	Prothrombin time, platelets, clot. retr.

When in doubt, obtain all these parameters–maintain the circulation and respiration and the data itself will take over the direction and sensible handling of the case. Everyone has his own order for doing these things and obtaining this data, and, of course, if along the route we come upon the answer, we can stop the pursuit of data. This suggests we should get that data first which is most likely on a statistical basis, to yield the clue to the unsolved clinical problem.

Case Report

A case report will be given to illustrate this approach:

I was called in consultation to see a 21-month-old girl who had become semicomatose during her 15 hours of hospitalization. The admitting diagnosis was pneumonia. On the telephone call from the physician, the initial check list of questions was followed revealing the following:

No history of head injury or seizures was known.

No drug ingestion prior to admission known.

The extent of pneumonia had not been assessed by x-ray nor function by blood gases.

No hepatic or endocrine disease was suspected.

The hematocrit was 35% and serum CO2 8 mEq per litre.

The low CO2 would have to be explained on the basis of severe metabolic acidosis or respiratory alkalosis. The implications of this were pursued by obtaining a serum ketone, blood sugar and salicylate level, followed by pH and pCO2. The salicylate level was 50 mg per 100 ml. and was done by the time (15 minutes) I arrived to examine the patient.

Examination of the patient revealed marked dehydration, semi-coma, rectal temperature of 101°F., slight resistance to neck flexion, respiratory rate of 60 per minute, rales in the right lung field, heart rate of 120 per minute, BP 80/60 mm Hg. The abdomen was soft. Petechiae were present in the antecubital fossae.

Proceeding to sort out the problems from head to toe:

Region	*Crucial Data*	*Management*
Head	– slight nuchal resistance.	– No L.P. because of petechiae and Rx same for pneumonia.
Neck	– —	
Lungs	– pCO2 30 mm Hg. (sl. resp. alkalosis) pneumonia and pneumothorax.	– I.V. antibiotics, later pleural drainage.
Heart	– B/P 80/60 P. 120	—
Abdomen	– —	—
Kidneys	– BUN 15 mg/100 ml	—
Blood	– ketones – neg; pH 7.23, CO2 Ct 13 mEq.	– I.V. fluids, NaHCO3.
	blood sugar 100 mg%	– I.V. fluids with Dextrose.
	serum salicylate – 50 mg%	– following serum & urine pH.
	prothrombin time 35 seconds.	– I.V. Mephyton® 10 mg.
	Hct. 35%, in 15 hours 28%	– blood transfusion

Therapeutic maneuvers were obvious as the data unfolded. The acidosis and salicylism were treated concomitantly with more adequate hydration, bicarbonate and potassium therapy, while monitoring the pH of the blood and urine. The anemia and clotting defect were managed with whole blood and Mephyton. The pneumothorax later required drainage because of tension. The patient was discharged on her 29th hospital day.

General Remarks About Treatment Of The Unconscious Patient

Treatment of the unconscious patient often is associated with ventilation and acid-base problems in one's efforts to maintain adequate perfusion, oxygenation and hydration until the patient's homeostatic mechanisms resume control. To assure adequacy of therapy in these areas, continuous nursing care, respirators with 24-hour maintenance and adequate monitoring equipment including pH and blood gases, minute volumes and cardiac activity are necessary.

Emergency treatment in two frequent causes of unconsciousness–head injury and barbiturate intoxication are non-specific, for example:

MacIver reported in 1958[1] that prompt tracheal intubation and support of the cardiorespiratory systems reduced the mortality rate from severe head injury with unconsciousness from 90% to 40%. No change in neurosurgical management was instituted. The routine and immediate tracheal intubation, aspiration and ventilation, if indicated, in the unconscious head injury patient was prompted by the frequent post-mortem findings of significant aspiration pneumonia, aspirated blood, and atelectasis.

With barbiturate intoxication, when the emphasis was shifted from that of analeptic therapy to adequate support of the cardiorespiratory systems, the mortality rate decreased from an average of 20% to less than 5%.[2,3]

In the management of the acidotic patient, one is limited by the diseased patient's lungs, and kidneys to make up for any errors in treatment. Two ways of minimizing this are: 1. monitor the pH at least as frequently as one does the hematocrit, pulse and blood pressure in the massive G.I. bleeder, and/or 2. perform peritoneal dialysis and let the serosa decide how much sodium, chloride, bicarbonate and potassium to keep or throw away–probably a better and safer approach than an "educated guess" at the acid-base problems.

Removal of the patient to a treatment center should be accomplished as soon as adequacy of the cardiorespiratory function is established in the event that adequate personnel and equipment are not locally available.

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Idiopathic Hypoparathyroidism: Report of a Case

JACK TATIROSIAN, M.D.†

Idiopathic hypoparathyroidism occurs sufficiently infrequently to warrant a case report. In 1958 Bronsky et. al.[1] reported a total of 50 cases in the English literature. In 1962 Dahl et. al.[2] commented on the addition of approximately 25 more cases.

The following case report is that of another patient with idiopathic hypoparathyroidism.

CASE REPORT

This 50-year-old housewife was hospitalized with a diagnosis of viral pneumonitis. She had never had a previous hospitalization. During routine evaluation of her illness, an abnormal prolongation of the Q-T interval was noted on the electrocardiogram (Fig. 1). Further investigation revealed the characteristic findings of hypoparathyroidism.

Past history and family history were unremarkable. There was no history of any previous thyroid surgery or irradiation of the neck. For the 8 months preceding admission, she had noted increased nervousness, shortness of breath, and paresthesias of the hands but had never experienced any seizures or eye difficulties. For menstrual irregularities, the patient had been given Ortho Novum® by her private physician approximately 1 year prior to admission.

Physical examination was within normal limits, except for the presence of râles in the posterior and lateral basilar segments of the left lower lobe. This subsequently cleared during her convalescence.

Chvostek's (Grade 1) and Trousseau's signs were easily demonstrable. The skin was dry but not scaly, and there were no noted abnormalities of the fingernails. Slit lamp examination revealed a small anterior sub-capsular opacity in the midperiphery of the right lens. Except for its location, it did not otherwise fit the pattern of a hypoparathyroid cataract.

The following laboratory data were obtained: CBC and urine were within normal limits except for a specific gravity of 1.010. Ca - 6.5 mg/100 ml., 6.6 mg/100 ml., 6.2 mg/100 ml. (Flame Photometer Method); P - 5.5 mg/100 ml., 5.6 mg/100 ml., 6.0 mg/100 ml. (Method of Fiske and Subbarow); alkaline phosphatase - 0.7, 0.8 Klein-Babson-Read units (normal 1.5 - 4.0); 24 hour urine for Ca and P (unrestricted diet) 46 mg/24 hours and 290 mg/24 hours, respectively; Na - 140 mEq/1, K - 4.5 mEq/1, Cl - 102 mEq/1, CO_2 - 25.5 mEq/1; total bilirubin 0.5 mg/100 ml.; direct bilirubin 0.1 mg/100 ml., SGOT - 16 units; amylase 80 units; total protein 6.4 gm/100 ml.; serum electrophoresis within normal limits; serology negative; BUN - 12 mg/100 ml.; creatinine clearance 131 ml/min; 15 minute PSP excretion 35%; urine culture negative; 3 hour glucose tolerance test:

fasting - 80 mg/100 ml.	2 hours - 80 mg/100 ml.
30 min - 132 mg/100 ml.	3 hours - 72 mg/100 ml.
1 hour - 116 mg/100 ml.	All urines were negative

Stool guaiac - negative for occult blood; stool stain for fat - small amount of neutral fat; chest X-ray - slight increase in bronchovascular markings; skull films - within normal limits without basal ganglia calcification; films of hands, feet and long bones within normal limits; tooth films - absence of lamina dura consistent with periodontoclasia; UGI and IVP series - within normal limits. EKG - prolongation of Q-T interval (0.48 seconds).

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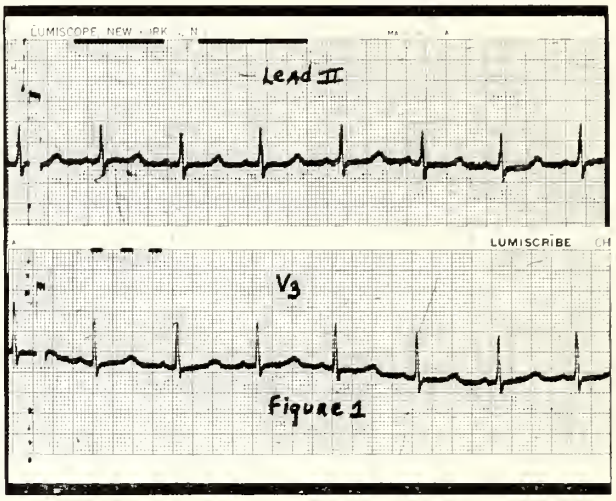


Figure 1

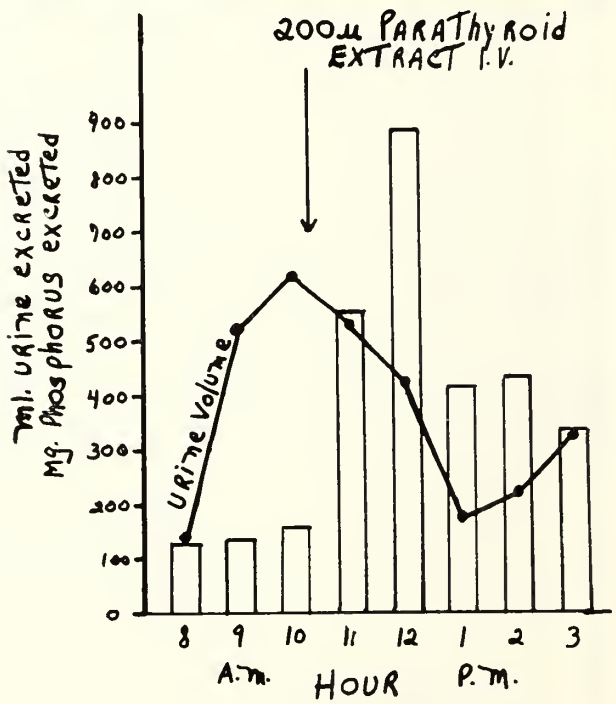


Figure 2

Intermediate PPD and Coccidioidomycosis intradermal tests - negative at 72 hours. Histoplasmosis - 12 mm induration at 72 hours; PBI - 10.4 u/100 ml. (patient on Ortho Novum®) - 17 - OH steroids - 4 mg/24 hours; 17 ketosteroids - 8.8 mg/24 hours. An Ellsworth-Howard test was performed in the fasting state with 200 units of parathormone* intravenously (Fig. 2). A normal control was not run simultaneously, nor was fluid

*Parathyroid injection, U.S.P. Eli Lilly and Company

restricted during the procedure. Objections to a positive interpretation of the test might be (1) absence of a normal control, (2) Non specific phosphorus diuresis resulting from increased fluid intake, (3) increased phosphorus excretion due to normal diurnal pattern. Nevertheless, the test was considered to exhibit a positive response with a significant phosphorus diuresis occurring during the first and second hours following parathormone administration.

After the appropriate laboratory studies were conducted, treatment was initiated with 100,000 units of Vitamin D and 4.8 grams of calcium lactate daily. The diet was unrestricted, but the patient was instructed to curtail milk and milk products. Weekly calcium and phosphorus determinations performed on an outpatient basis were 7.0 mg/100 ml., 7.6 mg/100 ml., and 8 mg/100 ml. of calcium; and 5.2 mg/100 ml., 5.8 mg/100 ml., 5.2 mg/100 ml of phosphorus. By the third week of treatment the serum calcium was 8 mg/100 ml. and the patient was symptomatically markedly improved. Chvostek's and Trousseau's signs were no longer demonstrable. Calcium lactate was increased to 8 grams daily and when last checked, the serum calcium was 9.2 mg/100 ml., the serum phosphorus 5.4 mg/100 ml. The patient was then temporarily lost to follow up for the following 6-week period.

Discussion

The case reported satisfies the criteria set forth by Drake et. al.[3] for the diagnosis of idiopathic hypoparathyroidism, in the absence of previous thyroid surgery: (1) Low serum calcium, (2) high serum inorganic phosphorus levels, (3) absence of roentgenologic signs of rickets and osteomalacia, (4) chronic tetany, (5) absence of renal insufficiency, steatorrhea, chronic diarrhea and alkalosis.

A positive Ellsworth-Howard test, in the absence of a family history, characteristic body habitus and bony changes of the hands and feet, is incompatible with the diagnosis of pseudohypoparathyroidism or pseudo-pseudohypoparathyroidism.

The etiology of hypoparathyroidism remains obscure. In the series of 13 cases reviewed by Drake et. al.[3] little of etiologic significance was noted. However, the onset of the disease, in several of the cases, coincided with acute infections. The demonstration of degenerative changes in the parathyroid glands during acute infections suggested that viral or bacterial agents might be significant factors.

Of the characteristic clinical findings in hypoparathyroidism, tetany, manifested by carpopedal spasm remains the most dramatic. It has been noted to occur in 78%,[4] 70%,[5] 76%,[1] 64%[6] of reported cases. Increased neuromuscular excitability resulting from low serum calcium levels may involve skeletal, smooth, or cardiac muscle. Tetanic equivalents manifested by numbness, paresthesias, muscle cramps and twitchings may be the only manifestations of this condition. Latent tetany may be elicited by the familiar Chvostek or Trousseau signs which have been cited by Steinberg and Waldron as being present in every case in which it was sought.[4] Involvement of the smooth muscle of the bronchi may produce an acute attack of bronchial asthma.[7] Death from asphyxia secondary to acute laryngeal spasm has been reported.[7]

Convulsions have been reported to occur in 52%,[4] 42%,[5] and 70%[1] of reported series. A significant number of patients particularly in the younger age groups were often mistakenly diagnosed as having idiopathic epilepsy. Papilledema, explained on the basis of cerebral edema[8] was found in 13.5% and 18% of the cases reviewed by Steinberg et. al.[4] and Bronsky[1] respectively. Mental disorders varying from frank phychosis to simple irritability and emotional instability have been reported to occur quite commonly.[9,10,11]

Involvement of ectodermal structures is often found as part of the clinical spectrum of hypoparathyroidism. Trophic changes consist of dry, scaly skin; brittle atrophic nails with transverse ridging; thin, patchy hair of the axilla, head, eyebrows and pubis. Monilial infections of the skin and nails are not uncommon. Delayed and defective dentition may occur if the hypoparathyroid state occurs prior to final tooth formation.

Cataract formation has been found in approximately half of the reported cases.[1,4] Typical lens changes consist of small discrete cortical opacities which may sometimes coalesce to form larger flakes. Eventually the lens becomes uniformly opaque.[12]

The most characteristic X-ray change, but by no means pathognomonic, is the presence of intracerebral calcifications in the region of the basal ganglia. Benett et. al. in studying a group of 88 patients with basal ganglia calcification noted 48 patients with idiopathic and 14 patients with pseudohypoparathyroidism.[13]

Finally, the electrocardiophic changes, by which the diagnosis of unsuspected hypoparathyroidism is often made, consists of prolongation of the Q-T interval. This prolongation usually involves only the segment between the QRS and T-wave proper and consists of a lengthened straight isoelectric line between these deflections.[14]

Summary

1. Idiopathic hypoparathyroidism is a rare disorder as judged by the relatively few reported cases.
2. A case fulfilling the criteria for the diagnosis of idiopathic hypoparathyroidism is reported.
3. The clinical features of this condition are discussed.

Acknowledgment

I would like to express my appreciation to Dr. Richard C. Wadsworth, Eastern Maine General Hospital, for his help and suggestions in the preparation of this manuscript.

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DEAN H. FISHER, M.D.
COMMISSIONER

State Of Maine

Department of Health and Welfare

Maine's Expanded Medical Care Program—Title XIX

Date July 22, 1966
To LOCAL MUNICIPAL OFFICIALS
From Dean Fisher, M.D., Commissioner, Department of Health and Welfare
Subject Medical Assistanee Program now in effect for all Public Assistance Recipients Under Titles 18 and 19 of Medicare

Effective as of July 1, 1966 new policies, procedures and methods of payment provide broader medical care in all public assistance categories. (Old Age Assistance, Aid to Families with Dependent Children, Aid to the Disabled and Aid to the Blind.) These programs replace all previous allowances in medical care programs now terminated or revised.

The purpose of this memo is to describe the new medical assistance programs and what they provide for the public assistance recipient:

1. Title 18 of the Social Security Act (P.L. 89-97) provides medical care for essentially all persons age 65 and over including Old Age Assistance recipients. Part A benefits are automatically available to these persons but Part B benefits are subject to the voluntary enrollment of the individual. Since this coverage meets the major medical needs of those whom the Kerr-Mills (Medical Assistance for the Aged) program formerly cared for, the Medical Assistance for the Aged program was discontinued June 30, 1966.
2. Title 19 of the Social Security Act (P.L. 89-97) provides this State with a favorable matching formula (Federal – State money) if we establish a medical assistance program primarily to meet the needs of public assistance recipients under the age of 65.

The following information should be your guide for utilization of these programs until such time as we can provide you with printed information. We have sent similar guides to all certified hospitals, physicians, other providers of services under Medicare. We have also notified all public assistance recipients regarding their new medical assistance coverage and how to apply when in need.

I. Recipients age 65 and Over. (Old Age Assistance)

Scope under Title 18 – known as Medicare.

Part A

Inpatient Hospitalization – up to 90 days per "spell of illness" and upon physician's recommendation. Payment for semi-private room accommodations will be made routinely.

Outpatient Diagnostic Services over a 20-day period recommended by the physician.

Services in an extended Care Facility will not be available until January 1, 1967. Until then the present nursing home program will continue with payments of $240 monthly (recently increased from $220).

Home Health Services – 100 visits within one year of discharge from a hospital or Extended Care Facility when provided by a certified agency in accordance with physician's plan for care following discharge from either facility.

Part B

Home Health Services – same as those under Part A but without the requirement of previous hospitalization.

Physicians Services – unlimited availability provided by medical doctor or osteopath licensed to practice in Maine. Such services, reimbursable at a "reasonable charge rate" may be provided in office, home, nursing home or elsewhere.

Other Medical Services – as recommended by physician such as diagnostic tests, x-ray or other types of therapy including costs of supplies and technicians services; drugs which physician administers and which cannot be self-administered; surgical dressings, casts, or splints; rental or medical equipment such as wheel chairs, hospital beds, iron lung; ambulance services to nearest hospital or other medical facility if essential; replacement of internal organs; artificial legs, arms, eyes, including replacements if required due to change in patient's condition.

Additional Benefits not covered under Part A or B but which Department will provide:

Necessary routine physical examinations.

Immunizations.
Outpatient hospital services including clinic care.
First three pints of blood.
Hospital coverage beyond 90 days for each "spell of illness" if necessary.
Home health services beyond 200 visits if medically necessary.

METHODS OF PAYMENT (Title 18)

For Part A of Medicare

The Associated Hospital Services is the designated agency through which Part A claims will be processed for all Title 18 beneficiaries, including Old Age Assistance recipients. For the OAA recipient, the Department will pay whatever deductibles and co-insurance charges may be appropriately incurred by the individual Old Age Assistance recipient.

For Part B of Medicare

The Department will pay the $3.00 monthly premium for these benefits on behalf of each Old Age Assistance recipient. This is accomplished by agreement with the Social Security Administration.

The Union Mutual Life Insurance Company is the designated agency through which Part B claims will be processed for all Title 18 beneficiaries, including Old Age Assistance recipients. All physicians services plus extended services noted above will be covered. The Department will also pay on behalf of the individual Old Age Assistance recipient any deductible or co-insurance charges involved in Part B.

Most payments will be made directly to the providers of medical services in behalf of the OAA recipients as for any other Social Security recipient. Such payments will be made by either the Associated Hospital Services or the Union Mutual and such reimbursement must be accepted as payment in full.

No payment for these services will be made by either the Social Security administration or the Department of Health and Welfare in those instances where other agencies have legal responsibility to provide medical services such as Workmen's Compensation cases or the Veteran's Administration.

Note: Spouses are not covered in the Department's plan for complementing Title 18 unless they are recipients of a public assistance grant in their own name.

II. Medical Assistance Program for those under 65 who receive Public Assistance

This program commonly referred to as *Title 19* covers those receiving Aid to Families with Dependent Children, Aid to the Disabled and Aid to the Blind.

The program will be administered directly by the Department and will not go through either the Associated Hospital Services or the Union Mutual Life Insurance for payment as does the program for Old Age Assistance recipients.

All recipients in these programs have been provided with a letter serving temporarily as their identification as well as informing them of the new coverage. They will soon receive identification cards good for a six-month period. However, because of the unpredictable factors in the determining of continuing eligibility for a public assistance payment, providers of medical care have been advised not to accept the card as final verification. This final verification of eligibility status will be made when the bill is received in central office and thus prompt billing is important to providers.

Coverage and Scope: In general all public assistance recipients will be eligible for all benefits listed under Parts A and B of Title 18 previously described plus the following additional services:

Necessary routine physical examinations.
Immunizations.
All outpatient hospital services including clinic care.
First three pints of blood.
Hospital coverage beyond 90 days if medically necessary.
Home Health Services beyond 200 visits if medically necessary and with no prior hospitalization required.

Payment – The Department of Health and Welfare will act directly as the fiscal agent for these recipients. The same rates and basis of payment will be used as by the two fiscal intermediaries for Parts A & B of Title 18 of Medicare.

It should be noted that *medicine* other than that provided during inpatient hospital care, or that necessarily administered by a physician on a non-in-patient basis, as a part of his services, are not covered by either Titles 18 or 19 or PL 89-97.

Payment by the Department in accordance with the above rates must be accepted as payment in full minus any deductions for individual insurance payments, and voluntary payments by the patient, family or other sources. No payment for these services will be made by either the Social Security Administration or the Department of Health and Welfare in those instances where another has legal responsibility to provide these medical services such as Workmen's Compensation cases or the Veteran's Administration.

Note: Adults or children in an AFDC household who are not included in the AFDC budget are *not* covered.

III. Out-of-State Coverage

It is not the intent of the Department to interrupt the usual patient-doctor relationship which may occur when a public assistance recipient lives on or near the State border and obtains his medical care either in New Hampshire or Canada when this is the usual established pattern. The Department will reimburse for such care by providers of services in accordance with its in-State practices when medical care is provided within 15 miles of the Maine-New Hampshire border, or 5 miles of the Maine-Canadian border. In the instance of the St. Stephen and Grand Falls Hospitals payment will be

made to these facilities on behalf of public assistance recipients only in emergency cases due to the fact that there are comparable hospital facilities in the United States within the 5 mile radius.

Medical services provided a Maine public assistance recipient during a temporary absence in another State will be comparable in scope to those available to the Maine resident and reimbursement will be similar to in-State payments.

If a public assistance recipient in Maine requires a type of medical care which is only available in an out-of-State medical facility (other than those hospitals excepted above in New Hampshire and Canada), approval prior to admission to the out-of-State medical facility must be obtained from the Department upon the request and justification by the attending physician.

IV. Unmet Medical Needs

The provisions described herein will result in considerable relief of financial worry for the many individuals who are now covered and will hopefully provide better health protection as well as remedial care. Even though the benefits will also be felt by municipalities where there is anticipated to be a reduction in the financial burden previously incurred for medical expenses now covered, there are still unmet medical needs which neither the Social Security Medicare under Title 18 nor the Department of Health and Welfare Medical Assistance Program under Title 19 can provide. These include:

Eye glasses or eye examinations.
Hearing aids or hearing examinations.
Prescription or patent medicines.
Orthopedic shoes or other supportive devices for the feet.
Cosmetic surgery to improve appearance unless related to a recent injury.
Routine dental care or dentures.
Private duty nursing or other private duty attendant.
Private room accommodations unless medically necessary.

We consider all of the above items important to providing comprehensive medical care but we consider prescription medicines and dental care (especially for children) our major medical needs still unmet by the broader medical services now available to recipients.

Until such time as we can extend the scope of medical services to include above unmet needs, municipalities will need to meet these and other unusual needs.

Note: The State Hospital Aid program for medically indigent under age 65 (non-public assistance recipients) has now been reduced to a $5.00 per diem allowance. The major part of State Hospital Aid appropriations have been transferred to the Title 19 Medical Care Program in which Federal matching of State money in a two for one ratio will permit better payment to hospitals in behalf of public assistance recipients.

For Your Information

We anticipate there will be many unanswered questions and many new situations needing clarification. Feel free to contact our Medical Unit in Augusta, Mr. Robert O. Wyllie, Supervisor, Extension 304.

ANNUAL MEETING DATES FOR YOUR 1967 CALENDAR

Maine Medical Association, June 11-13, 1967 at The Samoset, Rockland, Maine

American Medical Association, June 18-22, 1967, Atlantic City, New Jersey

"'Tranquilizer' is not a good word"[1]

"THIS classification is psychologically too seductive, pharmacologically too unspecific, and in terms of results not infrequently untrue."[2]

What is a tranquilizer? According to the 24th Edition of Dorland's Medical Dictionary[3] a tranquilizer is "an agent which acts on the emotional state, quieting or calming the patient without affecting clarity of consciousness."

Defining a drug by its effects, however, can be misleading. The same effects by which the dictionary defines a tranquilizer have sometimes been seen after administration of a sedative — or, for that matter, a placebo.

Ambiguous though the term may be, it appears to be here to stay. The 1966 edition of the Physicians' Desk Reference[4] lists 42 tranquilizers indicated for treatment of anxiety and apprehensive states.

'Tranquilizers' have differences in action, differences in effect

Although all tranquilizers are intended to calm anxious patients there are differences in their actions — and in their effects. They have been divided into three categories — the rauwolfia group, the 'minor' tranquilizers, and the phenothiazines.[5]

Although the tranquilizing effect of rauwolfia has been known for centuries, its use as an antipsychotic agent in current practice has diminished.[5]

A 'minor' tranquilizer is often prescribed to achieve more than one effect. Thus, besides being tranquilizers some of these compounds may be muscle relaxants, antihistaminics with some calming action, anticholinergic sedatives, or antispasmodics.[5]

The phenothiazines are considered 'major' tranquilizers because they alter psychotic behavior.[1] This classification may have done them more harm than good because it implies that the phenothiazines should be reserved for the more severely disturbed. This is not necessarily true.

The phenothiazines — and the problem of sedation

One of the problems of prescribing phenothiazines for ambulatory patients has been the fear that excessive sedation will impair the patient's ability to function. This, however, is less of a problem with some of the phenothiazines.

"Clinically they may be differentiated primarily in terms of their potency and the extent of their sedative effect, which appear to be inversely proportional. That is, the least potent, the one which is used in highest dosage, chlorpromazine, is the most sedative, while the reverse holds true for fluphenazine."[6]

In a recent report on various studies conducted over several years evaluating 360 patients treated for anxiety and stress with seven phenothiazines, this inverse relationship of potency to sedation was confirmed.[7] Also under consideration was the degree to which the particular phenothiazines neutralized anxiety (the angolytic index). Interestingly enough there was, again, an inverse relationship. The most sedative of the phenothiazines appeared to be the least active in neutralizing anxiety. Fluphenazine, one of the least sedative, on the other hand, was found to be most effective in relieving anxiety.[7]

RELATIVE SEDATIVE AND ANGOLYTIC INDICES OF PRINCIPAL PHENOTHIAZINES*

DRUG	SEDATIVE INDEX	ANGOLYTIC INDEX	BASED ON STANDARD DOSE OF
Chlorpromazine	100	15	25 mgs.
Triflupromazine	100	15	25 mgs.
Thioridazine	90	17	25 mgs.
Perphenazine	15	25	4 mgs.
Carphenazine	25	25	25 mgs.
Trifluoperazine	3.3	95	2.0 mgs.
Fluphenazine	3.5	100	2.5 mgs.

*adapted from Sainz[7]

Prolixin is therapeutically effective without excessive sedation

When used as a 'tranquilizer' in general medical practice, in many patients Prolixin (Squibb Fluphenazine Hydrochloride) suppresses anxiety, but not normal activity. These two features are of particular importance to patients who must be able to live and work without their normal daily activities being restricted.

Because of its longer duration of action, Prolixin, in doses of as little as one to three milligrams in adults, generally taken once a day, is effective in maintaining many patients free of their symptoms of anxiety and tension.

Contraindications: Do not use with high doses of hypnotics or in patients with subcortical brain damage. Use with caution in patients with a history of convulsive disorders. Severe reactions may occur in patients with idiosyncrasy to other centrally-acting drugs, and severe hypotension may occur in patients with special medical disorders, e.g. mitral insufficiency and pheochromocytoma.

Precautions: Effects of atropine, anesthetics and C.N.S. depressants may be potentiated. Hypotension may occur in patients undergoing surgery. Do not use epinephrine for treatment of the hypotensive reactions which may appear in patients on phenothiazine therapy.

Side Effects: Extrapyramidal reactions, allergic skin reactions, the possibility of anaphylaxis, drowsiness, visual blurring, dizziness, insomnia, nausea, anorexia, salivation, edema, perspiration, dry mouth, abnormal lactation, polyuria, hypotension, and jaundice and biliary stasis may occur. Routine blood counts are recommended to determine possible blood dyscrasias; if symptoms of upper respiratory infection occur, discontinue drug and institute appropriate therapy.

Available: 1 mg. tablets. Bottles of 50 and 500.

For full prescribing information, see package insert.

References: 1. Simpson, G.M.: Postgrad. Med. *39*:557, 1966. 2. Freyhan, F.A.: Am. J. Psychiat. *115*:577, 1959. 3. Dorland's Illustrated Medical Dictionary, ed. 24, Philadelphia, W. B. Saunders Co., 1965, p. 1603. 4. Physicians' Desk Reference, 1966, Oradell, N.J., 1965, p. 310. 5. Cohen, S.: Northwest Med.: *65*:197, 1966. 6. Detre, T., and Jarecki, H.: Connecticut Med. *25*:553, 1961. 7. Sainz, A.: Psychosomatics *5*:167, 1964.

PROLIXIN®
SQUIBB FLUPHENAZINE HYDROCHLORIDE

SQUIBB 'The Priceless Ingredient' of every product is the honor and integrity of its maker.

block
end
runs



LOMOTIL®

tablets/liquid

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride..... 2.5 mg.
(Warning: May be habit forming)
atropine sulfate .....0.025 mg.

tackles the problem of diarrhea directly

Effectiveness—Physiologic evidence indicates that Lomotil acts directly on the smooth muscle of the bowel to lower motility and control diarrhea. This action is unsurpassed in promptness and efficiency.

Convenience—Lomotil is available as small, easily carried, virtually tasteless tablets and as a pleasant, fruit-flavored liquid.

Versatility—The therapeutic efficiency, safety and convenience of Lomotil may be used to advantage alone or adjunctively in diarrhea associated with:

• Functional hypermotility • Acute infections • Malabsorption syndrome • Drug therapy • Gastroenteritis and colitis • Irritable bowel • Regional enteritis • Ileostomy • Ulcerative colitis • Food poisoning

For correct therapeutic effect
Rx correct therapeutic dosage

Dosage: The recommended initial daily dosages, given in divided doses until diarrhea is controlled, are:

Children: Total Daily Dosage

3-6 mo. . . ½ tsp. t.i.d. (3 mg.)
6-12 mo. . ½ tsp. q.i.d. (4 mg.)
1-2 yr. . . ½ tsp. 5 times daily (5 mg.)
2-5 yr. . . . 1 tsp. t.i.d. (6 mg.)
5-8 yr. . . . 1 tsp. q.i.d. (8 mg.)
8-12 yr. . . 1 tsp. 5 times daily (10 mg.)

Adults: 2 tsp. 5 times daily (20 mg.)
(or 2 tablets q.i.d.)

*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

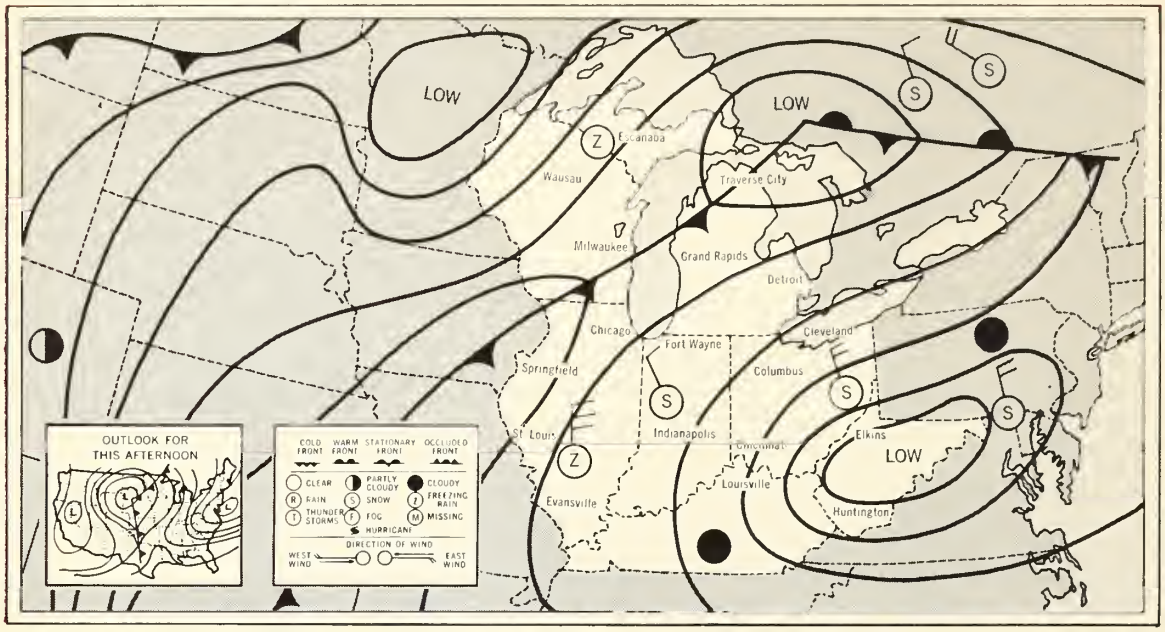
Precautions: Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is a Federally exempt narcotic preparation of very low addictive potential. Lomotil should be kept out of reach of children since accidental overdosage may cause severe respiratory depression. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. The subtherapeutic amount of atropine is added to discourage deliberate overdosage.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

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Tussagesic breaks up coughs, quickly clears stuffed and runny noses and relieves aches and pains. Provide coverage of the tough cold for up to 24 hours with just a single timed-release tablet dosed morning, midafternoon and at bedtime.

each

Tussagesic®

timed-release tablet contains:

Triaminic® (phenylpropanolamine hydrochloride 25 mg., pheniramine maleate 12.5 mg., pyrilamine maleate 12.5 mg.)	50 mg.
Dextromethorphan hydrobromide	30 mg.
Terpin hydrate	180 mg.
Acetaminophen	325 mg.

Dosage: Adults—1 tablet, swallowed whole to preserve timed-release feature, in morning, midafternoon and at bedtime. *Side effects:* Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets. *Precautions:* The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

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AMERICAN CANCER SOCIETY - MAINE DIVISION, INC.

Brunswick, Maine

Laryngectomees Make Good Employees

NEW YORK — Laryngectomees — persons who have had their voice box (larynx) removed surgically — make good employees, according to a new pamphlet published by the International Association of Laryngectomees (IAL).

Laryngectomees are working in all levels of manufacturing, industry, business, government, farming professions and public service industries, says the new pamphlet.

Tht IAL is sponsored by the American Cancer Society and is composed of more than a hundred "Lost Chord" or "New Voice" clubs whose members are laryngectomees dedicated to assisting in the total rehabilitation of new laryngectomees.

The pamphlet, now available at local ACS offices, lists a wide number of jobs performed by persons who have had their larynxes removed — most because of cancer. The jobs range from judge, drug clerk, cab driver, brick layer, secretary, mayor, waitress, steward, bus driver, fireman, salesman, chief of police, pharmacist, bartender to barber and many more.

It goes on to say that a person who has had the courage and perseverance to overcome a major handicap will make a fine employee. Customers will admire such a person and the employer can gain a great deal of good will by keeping him on the job.

There are about 27,000 persons in this country who are laryngectomees and the number is rising, says the pamphlet. About one out of five persons — according to a recent study, reported in the pamphlet, have not been retained in their jobs after their recovery. Attitudes towards these former patients have improved immensely, but some people are still prejudiced.

Cancer is neither contagious nor infectious, the pamphlet explains. "Superstition and 'hushed whisper' attitudes toward cancer have been largely erased. The scientific evidence that cancer is no more contagious than a heart attack or a broken leg has helped to remove the stigma that once surrounded cancer. The fear that other employees may discriminate against one who has recovered from cancer is outworn and has no place in today's world of great progress in science and medicine."

The larynx is located on top of the windpipe, right in front of the esophagus (food pipe). When the larynx has to be removed, the IAL pamphlet explains, in order to provide air to the lungs, the surgeon creates an opening at the base of the throat and attaches the windpipe to it.

"The laryngectomee can breathe, but he cannot expel air from the lungs for speaking, because there is no connection between the lungs and the mouth or nose."

He is taught to force air into the upper esophagus and pharynx and to expel it immediately, causing a low-pitched sound. This sound, which is shaped into words by use of the tongue, lips, teeth and cheeks as in normal speech, becomes the laryngectomee's voice, states the pamphlet.

Employers interested in further facts are urged to contact their local ACS offices. A Lost Chord member will call on them.

American Cancer Society News Service Release

Maine Heart Association Notes

116 State Street, Augusta, Maine

Clinical Significance and Consequences of Labile Hypertension

. . . "labile hypertension is present if the patient usually has casual blood pressure readings in the normal range and occasional readings above the normal range, that is above 150/90 mm. Hg.

Many investigators have noted the tendency of labile hypertension to progress to sustained hypertension.

Despite some empty talk to the contrary and despite some exceptions, elevated diastolic blood pressure significantly affects prognosis: The higher the blood pressure, the worse the prognosis. Morbidity and mortality are caused by damage to the heart, brain and kidneys. . . . At the present it would be well to study the hemodynamics of the patient being evaluated because of blood pressure elevation. If cardiac output and peripheral resistance could be determined, more certain indications for therapy might be made and more certain knowledge of his future might be at hand.

Because of the studies previously mentioned which show that many people who have transient hypertension will ultimately develop persistent sustained hypertension with vascular and organ damage and early death, it would seem important to begin antihypertensive therapy as more and more diastolic readings of 100 mm. or over are recorded."

Submitted by Jacob B. Dana, M.D., Maine Heart Association.

Reference: Housel, Edmund L., M.D., American Journal of Cardiology, Vol. XXXII, pages 617-620, 1966.



Homecoming is great . . .

if you have Blue Shield for doctor bills

Blue Shield often makes the difference between a harried homecoming and a happy one. Last year, Blue Shield paid more than a *billion* dollars in doctor bills on behalf of its 53 million members. And Blue Shield pays out more of the dollar in benefits because *nobody* in the voluntary medical prepayment field has lower operating expenses.

Employers value the convenience of Blue Shield since the doctor is normally paid direct—leaving little or no red tape or paperwork. Small wonder 7 out of 10 of America's largest industrial corporations favor Blue Shield for their employees.

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against the usual gram-negative urinary pathogens

Why use five...where **one** will do?



In a recent 217-patient hospital study,[1] urinary tract infections were treated with a variety of widely prescribed antimicrobial agents including: a sulfonamide (40 patients), chloramphenicol (20 patients), nitrofurantoin (33 patients), nalidixic acid (30 patients), tetracycline (27 patients), colistimethate sodium (22 patients)...and 2 combinations of 5 agents each (45 patients). The 2 combinations were selected to afford maximal theoretical antibacterial coverage against the usual urinary pathogens. They were (1) tetracycline, chloramphenicol, nitrofurantoin, ristocetin and polymyxin B; and (2) tetracycline, chloramphenicol, erythromycin, nitrofurantoin and colistimethate sodium.

This clinical study shows that the two combinations of antibiotics were not superior to some of their single components. The authors point out that antibiotic antagonism often negates theoretical advantages of multiple therapy. Coly-Mycin Injectable (colistimethate sodium) was one of the single components that was shown to be equal to the combinations and eradicated bacteriuria in two-thirds of the patients.

Theoretical choice of multiple antibacterial therapy has been shown to be no more effective than one well-chosen agent which also offers least patient exposure to possible side reactions, toxicities, allergic manifestations and higher drug costs.

1. McCabe, W. R., and Jackson, G. G.: New England J. Med. 272:1037, 1965.

in gram-negative urinary tract infections often the single well-chosen agent



Coly-Mycin® Injectable
(colistimethate sodium)

Indications: Especially indicated for the treatment of severe acute and resistant chronic urinary tract infections due to sensitive strains of gram-negative organisms. Also indicated in respiratory tract, surgical, wound and burn infections and in septicemia due to sensitive organisms. Particularly indicated when any of these infections are caused by sensitive strains of *Pseudomonas aeruginosa.*

Adverse Reactions: Occasional reactions such as circumoral paresthesias, tingling of the extremities, pruritus, vertigo or dizziness may occur. Reduction of dosage may alleviate symptoms. Therapy need not be discontinued, but such patients should be observed with extra care.

Warning: Patients should be cautioned not to drive vehicles or use hazardous machinery while on therapy.

Precautions: In cases of impaired or suspected renal impairment, use with greater caution and reduce dosage in proportion to extent of impairment. Transient elevations of BUN have been reported. As a routine precaution, appropriate blood studies should, therefore, be made during prolonged therapy.

As with all polypeptides, the possibility of muscular weakness, including apnea, due to inadvertent overdosage or normal dosage in the presence of impaired renal function, should not be overlooked. In cases of apnea, medication should be promptly discontinued and assisted respiration given until serum levels fall and normal breathing is restored.

Other antibiotics, such as kanamycin, streptomycin, dihydrostreptomycin, polymyxin, and neomycin, may also have varying neurotoxic or nephrotoxic potential. They should be used with great caution concomitantly with Coly-Mycin Injectable (colistimethate sodium).

For deep intramuscular injection only.

Dosage: By the I.M. route only, in 2 to 4 divided doses ranging from 1.5 to 5 mg./Kg./day (0.7 mg. to 2.3 mg./lb./day). Average adult dose is 2.5 mg./Kg./day (1.1 mg./lb./day). In the presence of bacteremia, septicemia, or other serious infection, greater than average doses may be required; however, maximum daily doses should not exceed 5 mg./Kg. (2.3 mg./lb.) where renal function is normal.

Not recommended against Proteus.

Colistin is also available (as colistin sulfate) in: Coly-Mycin® Pediatric for Oral Suspension (not for systemic use), and Coly-Mycin® Otic with Neomycin and Hydrocortisone.

Full information is available on request.

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in chronic illness

B and C vitamins are part of therapy: An imbalance of water-soluble vitamins and chronic illness often go hand in hand. STRESSCAPS capsules, containing therapeutic quantities of vitamins B and C, are formulated to meet the increased metabolic demands of patients with physiologic stress. In chronic illness, as with many stress conditions, STRESSCAPS vitamins are therapy.



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FOR PROFESSIONAL RECOMMENDATION ONLY

Each capsule contains:	
Vitamin B_1 (as Thiamine Mononitrate)	10 mg
Vitamin B_2 (Riboflavin)	10 mg
Vitamin B_6 (Pyridoxine HCl)	2 mg
Vitamin B_{12} Crystalline	4 mcgm
Vitamin C (Ascorbic Acid)	300 mg
Niacinamide	100 mg
Calcium Pantothenate	20 mg

Recommended intake: Adults, 1 capsule daily, for the treatment of vitamin deficiencies. Supplied in decorative "reminder" jars of 30 and 100; bottles of 500.

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New England Postgraduate Assembly

Statler Hilton Hotel. Boston. Massachusetts—Jan. 17-19, 1967

TUESDAY, JAN. 17

BALLROOM ASSEMBLY

A.M.

7:30 CONTINENTAL BREAKFAST

8:00 MOVIE: DNA — THE STORY OF LIFE
Discussor: William H. Baker, M.D., Boston, Mass.

9:00 BREAST CANCER
Moderator: Oliver Cope, M.D., Boston, Mass.
Panel: Robert Ravdin, M.D., Philadelphia, Pa., Henry Kohn, M.D., Boston, Mass., Eleanor D. Montague M.D., Houston, Texas and Benjamin Castleman, M.D., Boston, Mass.

10:20 PANEL ON POSTGRADUATE EDUCATION
Moderator: George Dunlop, M.D., Worcester, Mass.
Panel: Earle Wilkins, M.D., Daniel D. Federman, M.D., George J. Robertson, M.D. and Norman Stearns, M.D., Boston, Mass.

12:30 LUNCHEON: PARLORS A&B
TRAUMATIC SURGERY OF FACE AND SKIN
Speaker: Bradford Cannon, M.D., Boston, Mass.

P.M.

2:00 PREOPERATIVE MEDICAL EXAMINATION
Moderator: W. Allan Tisdale, M.D., Burlington, Vt.
Introduction: W. Allan Tisdale, M.D.
Panel: Burton S. Tabakin, M.D., George W. Welsh, III, M.D., Thomas C. Gibson, M.D., Burlington, Vt. and W. Allan Tisdale, M.D.

3:20 THE PATHOPHYSIOLOGY OF THE ALCOHOLIC NEUROLOGICAL DISORDERS
Kurt Isselbacher, M.D., Boston, Mass.
Maurice Victor, M.D., Cleveland, Ohio

8:00 SUBVERSIVE ACTIVITIES OF THE COMMUNISTS IN THIS COUNTRY
Speaker: Dr. Fern C. Stukenbroeker, Washington, D.C.

WEDNESDAY, JAN. 18

BALLROOM ASSEMBLY

A.M.

7:30 CONTINENTAL BREAKFAST

8:00 MOVIE: DIAGNOSTIC SIGNIFICANCE OF VENOUS PULSE, BY DR. PAUL WOOD

9:00 THE MEDICAL AND SURGICAL ASPECTS OF ACQUIRED VALVULAR HEART DISEASE
Moderator: W. Gerald Austen, M.D., Boston, Mass.
Panel: Roman DeSanctis, M.D. and Charles Sanders, M.D., Boston, Mass.

10:20 SURGICAL TREATMENT OF VASCULAR DISEASE
Moderator: Michael DeBakey, M.D., Houston, Texas
Panel: Allan Callow, M.D., John Mannick, M.D. and Robert Linton, M.D., Boston, Mass.

12:30 LUNCHEON: PARLORS A&B
MYCO BACTERIAL DISEASE: CHILD AND ADULT
Speaker: Kenneth Bird, M.D., Boston, Mass.

P.M.

2:00 SPARE PARTS AND TRANSPLANTS
Moderator: David Hume, M.D., Richmond, Va.
Panel: Anthony P. Monaco, M.D. and Joseph Murray, M.D., Boston, Mass.

3:20 PRESIDENT'S PROGRAM FOR STROKE, HEART AND CANCER CENTERS
Michael DeBakey, M.D.

8:00 PUBLIC RELATIONS
Professional Staff from the AMA, Chicago, Ill.

THURSDAY, JAN. 19

BALLROOM ASSEMBLY

A.M.

7:30 CONTINENTAL BREAKFAST

8:00 MOVIE: ABORTION AND THE LAW
Discussor: John Grover, M.D., Boston, Mass.

9:00 PANEL ON SEX EDUCATION
Moderator: Thomas J. Boag, M.D., Burlington, Vt.
Panel: Mary Jane Gray, M.D., J. Ward Stackpole, M.D. and Victor E. Chase, M.D., Burlington, Vt.

11:00 RECOGNITION AND TREATMENT OF MENTAL RETARDATION
Moderator: Mary Louise Scholl, M.D., Boston, Mass.
Panel: Richard L. Naeye, M.D., Burlington, Vt., Mary L. S. Efron, M.D., Randolph K. Byers, M.D. and Murray Sidman, M.D., Boston, Mass.

12:30 LUNCHEON: PARLORS A&B
THE USE OF ALLOPURINOL IN GOUT AND HYPERURICEMIA
Speaker: Stephen Goldfinger, M.D., Boston, Mass.

GEORGIAN ROOM

P.M.

2:00 THE ABORTION PROBLEM
John Grover, M.D.

3:00 HEART DISEASE IN INFANCY
Allan Goldblatt, M.D., Boston, Mass.

3:30 CLINICOPATHOLOGICAL CONFERENCE

4:30 ADJOURNMENT

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Secretary, Donald L. Anderson, M.D., Lewiston

AROOSTOOK

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County Society Notes

CUMBERLAND

A meeting of the Cumberland County Medical Society was held at the Holiday Inn in Brunswick, Maine on October 20, 1966 with fifty-nine members and guests attending.

The President, Alvin A. Morrison, M.D. announced invitations to attend a meeting of the Maine Dietetic Society, a symposium on Pulmonary Disease at the Osteopathic Hospital of Maine, and a meeting of the Councils of County Medical Societies. A letter was read from Mr. Kendall H. Dunbar of the Industrial Accident Commission thanking the membership for their cooperation in examining persons covered by Workmen's Compensation.

Dr. Morrison announced that the "Lederle" meeting would be held in co-sponsorship with the Maine Chapter of the American Academy of General Practice on October 19, 1967 with Drs. Morrison, MacVane, Bacastow and Charles Geer arranging the program.

Resolutions were delivered on the deaths of Daniel M. Rowe, M.D. and Frederick W. Skillin, M.D.

Charles R. Glassmire, M.D., Councilor of the First District, reported on a variety of items: that the House of Delegates in June had defeated the proposal by John R. Lincoln, M.D. that only members of the Council become members of the Public Relations Committee and that the state dues would be increased. He also mentioned that a medical school had not been declared feasible for Maine; that various groups are forming to organize Home Health Care under Title 19 of Medicare and that the State Street Trust Company in Boston is acting as a billing agent for Massachusetts physicians under Medicare. Edward G. Asherman, M.D. emphasized that when billing a lawyer for a medical report, itemize it as such, rather than "for professional services."

Paul C. Marston, M.D. announced that Lloyd G. Davies, M.D. had been elected as a national director of the American Academy of General Practice. Daniel F. Hanley, M.D., Executive Director of the Maine Medical Association, announced a need for appointment of committees to assist in liaison and in utilization review for long-term health care facilities. The membership voted to authorize the officers and executive committee of the Society to appoint such committees as is necessary. Albert Aranson, M.D. pointed out that these facilities will be very carefully regulated and will not include custodial care, although legislation for custodial care has been proposed.

Dr. Sylvester announced a new system of telephoned reports from physicians being utilized by insurance companies, and that the officers of the Society are working on a revision of the Bylaws. There was discussion of the fact that the South Portland Visiting Nurse Association is not recognized by Medicare with no solution to the problem being offered. The meeting was adjourned after announcement that the next meeting would be devoted to medical-legal matters.

STANLEY B. SYLVESTER, M.D., *Secretary*

KENNEBEC

The Kennebec County Medical Association met at the Holiday Inn in Waterville, Maine on October 20, 1966. The meeting was called to order by the President, Allan J. Stinchfield, M.D.

It was moved by Robert L. Ohler, M.D. that dues be raised to $35.00 a year and was seconded by Dr. Crawford. The motion was defeated. John F. Reynolds, M.D. then moved that the dues stay at $10.00 a year which was approved.

A nominating committee was appointed to prepare a proposed slate of officers for 1966-1967 by Dr. Stinchfield con-

sisting of the following members: Drs. John D. Denison, John F. Reynolds, Brinton T. Darlington and M. Tieche Shelton.

Dr. Ohler reminded the Association that at Togus they were making a study of delirium tremens and requested that any Veteran with the DT's be referred to Togus for evaluation. He promised immediate admission providing the patient had not been treated with Thorazine® or Phenobarbital prior to admission.

Alta Ashley, M.D. stated that things were ready for the Diabetes Week.

George W. Hyatt, M.D. of Washington, D.C. was the guest speaker. He presented a very concise straightforward presentation of the conservative treatment of the low back.

John C. Patterson, M.D. announced that he would obtain Dr. Robey to speak on human behavior and requested that members of the Androscoggin County Medical Association be invited for the December meeting at the Augusta State Hospital.

LUCIEN F. VEILLEUX, M.D., *Secretary*

WASHINGTON

Twenty-nine members and guests were present at a meeting of the Washington County Medical Society which was held on October 31, 1966 at the Dennys River Inn in Dennysville, Maine.

The President, Robert G. MacBride, M.D. introduced George E. Sullivan, M.D., President of the Maine Medical Association. Dr. Sullivan spoke of the necessity of contacting the members of the various political parties, in reference to bills that would be pending before the next Legislature and discussed other matters regarding the Society.

James C. Bates, M.D., Councilor of the Fifth District, mentioned the work of the Council and the amount of work that had to be done at each Council meeting. Both he and Dr. Sullivan said that much of the excellent position of the State Medical Association was due to the efforts of the Executive Director, Daniel F. Hanley, M.D.

Dr. MacBride introduced Mr. Richard F. Nellson, Executive Director of the Associated Hospital Service of Maine. Mr. Nellson covered the various aspects of the present Blue Cross-Blue Shield program and particularly the effect of the new Medicare plan. In the absence of Mr. Robert Cash of the Union Mutual Life Insurance Company, Mr. Nellson covered the Part B portion of Medicare.

The Society has been holding monthly meetings at the Dennys River Inn for the past four months. The attendance has usually been small but the discussion has been quite active and has helped to keep the Society on an active basis and to help lay the groundwork for this important meeting.

KARL V. LARSON, M.D., *Secretary*

New Members

ANDROSCOGGIN

Jan Knoppers, M.D., St. Mary's Gen. Hosp., Lewiston
James V. I. O'Sullivan, M.D., 376 Main St., Lewiston
Thomas F. Shields, M.D., 300 Pine St., Lewiston

CUMBERLAND

Sara K. Cope, M.D., 32 Deering St., Portland
Howard R. Deming, M.D., Maine Medical Ctr., Portland
Carolina R. Ines Campomanes, M.D., 144 State St., Portland
William E. Loring, M.D., 144 State St., Portland
Erwin G. Rerrick, M.D., Box C, Pownal
Ronald A. Swanson, M.D., Regional Mem. Hosp., Brunswick

HANCOCK

Walton P. C. Clason, M.D., 50 Union St., Ellsworth
Randall H. Silver, M.D., Maine Coast Mem. Hosp., Ellsworth

KENNEBEC

James C. Hayes, M.D., 6 E. Chestnut St., Augusta

News, Notes and Announcements

Clinic Schedule — 1966
Department of Health and Welfare
Division of Maternal and Child Health
Including Services for Crippled Children
(By Appointment Only)

Pediatric Clinics

Bangor — Eastern Maine General Hospital
1:30 p.m.: Oct. 28, Nov. 18, Dec. 23

Cardiac Clinics

Bangor — Eastern Maine General Hospital
9:00 a.m.: Oct. 28, Nov. 4, 18, Dec. 9, 23
Portland — Maine Medical Center
9:00 a.m.: Every Friday (holidays excepted)

Cystic Fibrosis Clinics
(In conjunction with the CMGH and MMC)

Portland — Maine Medical Center
9:00 a.m.: Oct. 18, Nov. 15, Dec. 20

Orthopedic Clinics

Augusta — Augusta General Hospital
1:00 p.m.: Dec. 22
Bangor — Eastern Maine General Hospital
Half-day Sessions: 1:00 p.m.: Oct. 27, Dec. 22
Lewiston — Central Maine General Hospital
9:00 a.m.: Oct. 21, Nov. 18, Dec. 16
Portland — Maine Medical Center
9:00 a.m. (In conjunction with MMC): Nov. 14, Dec. 12
Rumford — Community Hospital
1:30 p.m.: Dec. 21

Clinics for Mentally Retarded Preschool Children

Waterville — Thayer Hospital
9:00 a.m.: Oct. 19, Nov. 2, 16, 30, Dec. 7, 21
Lewiston — Central Maine General Hospital
9:00 a.m.: Nov. 14, Dec. 12
Lewiston — St. Mary's General Hospital
9:00 a.m.: Oct. 24, Nov. 28, Dec. 26

State of Maine Board of Registration of Medicine
Secretary — George E. Sullivan, M.D.
Waterville, Maine

Physicians Licensed to Practice Medicine and Surgery in the State of Maine
July 12-14, 1966

Through Examination

Manoochehr Aboozia, M.D., 359 Ashmont St., Boston, Mass.
Kazim S. Apaydin, M.D., Box C, Pownal, Me.
Kurt Aterman, M.D., Children's Hosp., Buffalo, N.Y.
Ivan G. Dimich, M.D., 665 New York Ave., Brooklyn, N.Y.
Manfred M. F. Ernesti, M.D., 15 Cedar St., Braintree, Mass.
Abdollah S. Gashgai, M.D., 300 Main St., Lewiston, Me.
Bela Gavaller, M.D., 184 Pine Grove Ave., Newton Lower Falls, Mass.
Harry L. Ioachim, M.D., Columbia Univ., New York, N.Y.
Massih H. Kish, M.D., St. Mary's Hosp., Passaic, N.J.
Mario Lecuona, M.D., Lakeville Hosp., Middleboro, Mass.
Richard S. W. Lugg, M.D., 12 Fox Meadow Lane, Wayland, Mass.
Robert F. McGann, M.D., 7 St. Catharines Rd., Broxbourne, Herts, England
Milorad M. Milic, M.D., 836 North 5th St., Steubenville, Ohio
Carmosina C. do Nascimento, M.D., 5508 Little Flower Ave., Cincinnati, Ohio
Thomas W. Samuelsen, M.D., 194 Main St., Lincoln, Me.
Sami A. Sfeir, M.D., 162 Larkmont Dr., Elyria, Ohio
Sarjit Singh, M.D., 5260 Kent St., Halifax, N.S.
Mahmood N. Tafreshi, M.D., Queens Hosp. Ctr., Jamaica, N.Y.
Joseph Vydas, M.D., Box 926, Bangor, Me.
Virgil G. Wright, M.D., Down East Comm. Hosp., Machias, Me.

Through Reciprocity

Louis H. Averbach, M.D., 5245 Oxford Ave., Philadelphia, Pa.
Harry W. Bennert, Jr., M.D., Maine Medical Ctr., Portland, Me.
Dale C. Bouton, M.D., 2 High St., Dexter, Me.
Richard W. Britt, M.D., 135 Adam St., Tonawanda, N.Y.
Hagop Halladjian, M.D., Rumford Comm. Hosp., Rumford, Me.
James C. Hayes, M.D., 6 E. Chestnut St., Augusta, Me.
Warren C. Hazelton, M.D., 2 E. Main St., So. Paris, Me.
Louis E. Jorel, M.D., 12 Thomas Ave., Caribou, Me.
Richard A. Kemp, M.D., 5 Surf Rd., Cape Elizabeth, Me.
Bharat B. Kumar, M.D., Galax Medical Ctr., Galax, Va.
George P. Lord, M.D., Strong Mem. Hosp., Rochester, N.Y.
William E. Loring, M.D., 144 State St., Portland, Me.
George V. A. MacDonald, M.D., Ashland, Me.
Stuart W. McGuire, M.D., Portland, Me.
Enid L. Melville, M.D., RFD, Machias, Me.
Buell A. Miller, M.D., Reef Rd., Cape Elizabeth, Me.
JoAnn M. H. Miller, M.D., Reef Rd., Cape Elizabeth, Me.
Pierre E. Provost, M.D., 48 Green St., Augusta, Me.
Erwin G. Rerrick, M.D., Box C, Pownal, Me.
Thomas F. Shields, M.D., 300 Pine St., Lewiston, Me.
Frank R. Smith, M.D., North Haven, Me.
Bella S. Strauss, M.D., Maine Coast Mem. Hosp., Ellsworth, Me.
Philip G. Whitney, M.D., 30th Field Hosp., APO, New York, N.Y.

Book Review

Surgical Diseases of the Chest, Edited by Brian Blades, Ed. 2. Published by the C. V. Mosby Company, St. Louis, 1966. 687 pages with 295 illustrations. Price $25.00.

The second edition of "Surgical Diseases Of The Chest" represents the collaborative effort of twenty distinguished thoracic surgeons, most of whom are either direct or indirect disciples of Evarts Ambrose Graham to whose memory the volume is dedicated. Notable exceptions to the above surgical lineage are the contributions on cardiac and great vessel surgery by Effler of the Cleveland Clinic, Harken of the Peter Bent Brigham, and DeBakey and Cooley of Baylor. There are also contributions by Dr. Kelser, a cardiologist, and Dr. Coakley, Professor of Anesthesiology at George Washington University. This, of course is within the avowed purpose of the book as stated in the preface, namely: "A book useful not only to surgeons, but also to medical practitioners and medical students who are interested in what can be accomplished by the surgical treatment of various diseases of the chest."

Not the least valuable feature of the textbook is the extensive list of suggested references at the end of each chapter. These seem to have been picked with both the historical and practical importance of the contributions in mind. Almost each chapter list of references has been up-dated since the last edition.

"Surgical Diseases Of The Chest" does not try to be an encyclopedia of the subject it undertakes but as stated in the preface by Dr. Blades, "Only the essential technical principles for standardized and accepted operations will be included. More detailed descriptions will be undertaken for new operations not included in older textbooks." This still makes it necessary for the student to refer to the older textbooks of thoracic surgery or to the specific articles if for instance he wishes to discover the technique of the Schede type of thoracoplasty or the details of an Eloesser flap procedure. Dr. Blades, who is the Louis Saltz Professor of Surgery and chairman of the Department of Surgery at the George Washington University School of Medicine in Washington, D.C. has used his editorial know-how not only with regard to the contributors he has chosen for the specific chapters, but also by omitting to mention operations or therapy with which, I presume, he does not agree.

Certainly this is an excellent textbook of thoracic surgery and although at these prices, I cannot say that every physician should have one. I believe that it should be available to every physician and surgeon in his hospital library and would make a valuable addition to any surgeon's personal library.

WILLIAM L. MACVANE, JR., M.D., Portland, Maine

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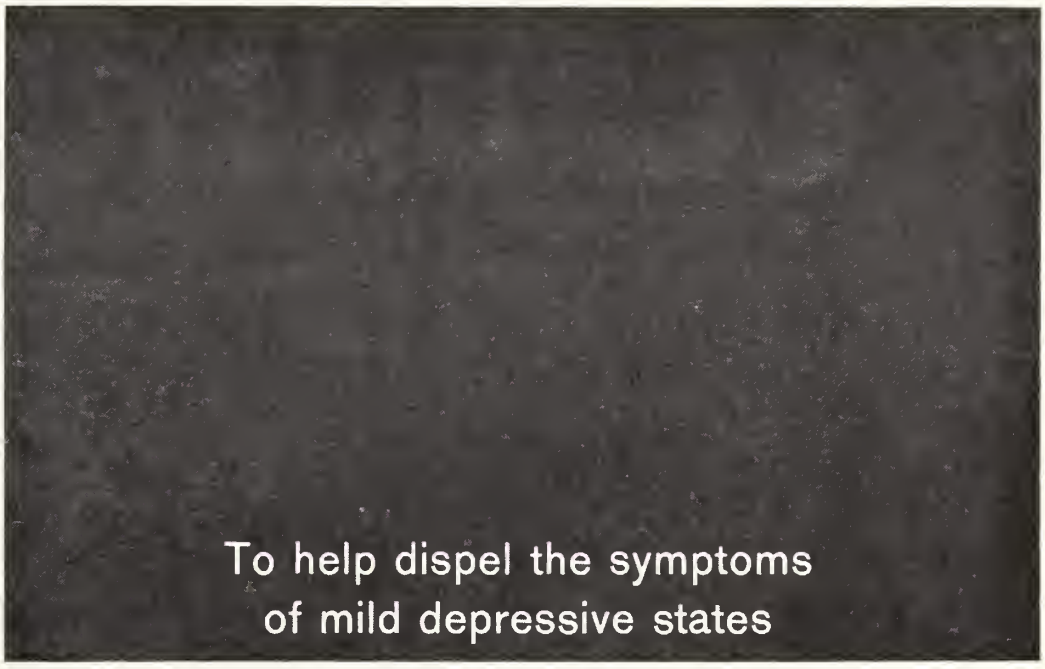
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The following is a brief precautionary statement. Before prescribing, the physician should be familiar with the complete prescribing information in SK&F literature or *PDR.*

Precautions: Use with caution in patients hypersensitive to sympathomimetics or barbiturates and in coronary or cardiovascular disease or severe hypertension. Do not use in patients taking MAO inhibitors. Excessive use of the amphetamines by unstable individuals may result in a psychological dependence; in these instances, withdraw the medication. Use cautiously in pregnant patients, especially in the first trimester. *Side effects:* Insomnia, excitability and increased motor activity are infrequent and ordinarily mild.

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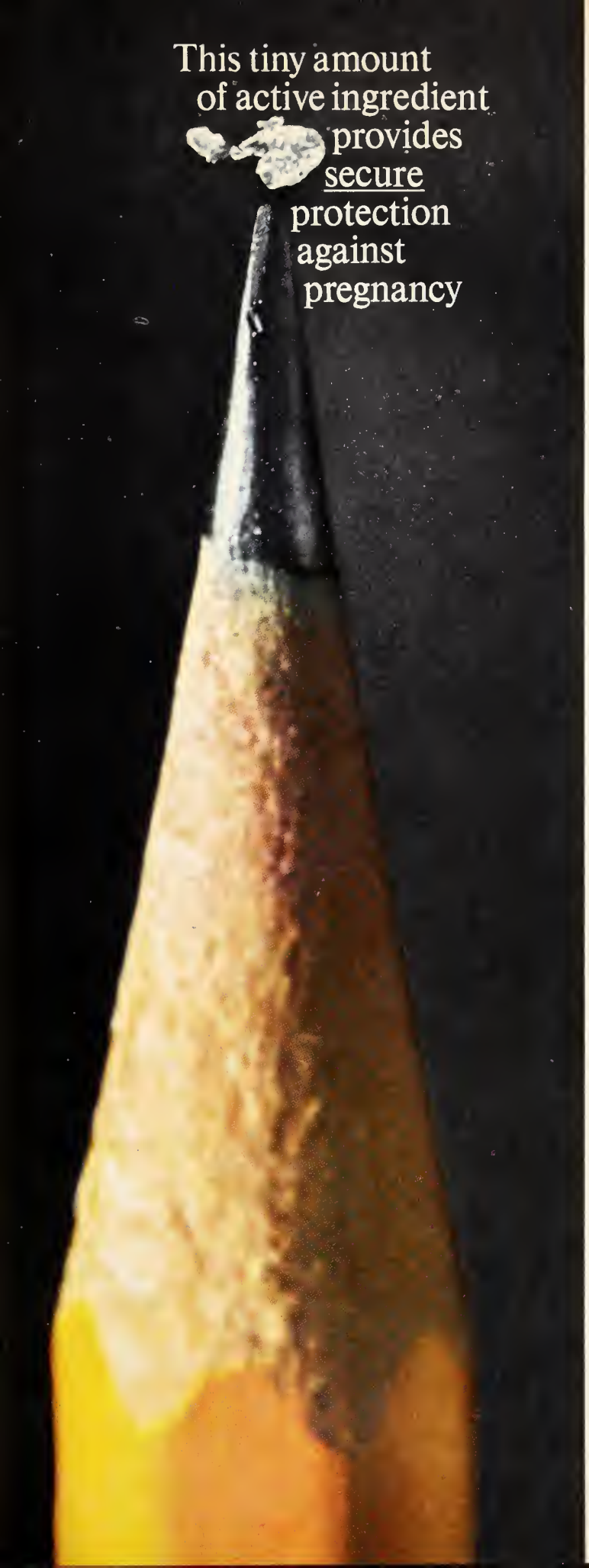
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Contraindications—Mammary or genital carcinoma should be ruled out prior to administering Ovulen. Undiagnosed vaginal bleeding is also a contraindication to Ovulen use. A history of thrombophlebitis or pulmonary embolism, or both, is a contraindication to the use of Ovulen, unless its use is judged by the physician to be necessary despite the possible risk. Ovulen should not be used in women with suspected or overt liver dysfunction or disease. Ovulen is contraindicated in pregnant and nursing women and in patients with a history of cerebrovascular accident.

Warnings—Medication should be discontinued pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. Medication should be withdrawn if examination reveals papilledema or retinovascular lesions. Since the safety of Ovulen therapy in pregnancy has not yet been established, it is recommended that, in a patient who has missed two consecutive menstrual periods, pregnancy be ruled out before oral contraceptive therapy is continued.

Precautions—Because Ovulen may aggravate a tendency toward fluid retention in some patients, it should be administered cautiously to patients with a history of renal or cardiovascular disease (including hypertension), asthma, epilepsy or migraine. Any possible influence of long-term Ovulen therapy on pituitary, adrenal cortical, ovarian, hepatic or uterine functions requires further study. Oral contraceptives also should be administered cautiously to diabetic patients since a decrease in glucose tolerance has been observed in some patients taking these drugs. Patients on Ovulen may occasionally show abnormal glucose tolerance tests, but this does not necessarily indicate the presence of diabetes. Significant increases in platelet count, prothrombin and proconvertin tests, plasma thrombotic activity and plasma proteolytic activity have been reported.

Since estrogens may affect results of serum protein bound iodine and other thyroid function tests, these tests should not be considered definitive until Ovulen therapy has been discontinued for at least sixty days. Adrenal steroid serum levels and excretion may be affected by estrogens; the Metopirone® (SU-4885) test of pituitary-adrenal function may also be depressed. Abnormalities in hepatic function tests have also been reported, including some interference with dye excretion by the liver. This interference may give rise to Bromsulphalein® retention and jaundice in susceptible individuals. Serious liver dysfunction should be ruled out before continuing Ovulen administration when abnormalities in liver function tests occur.

Patients with a history of psychic depression should be observed carefully during treatment with oral contraceptives, and such treatment should be discontinued if depression recurs to a serious degree. Pre-existing fibroids may increase in size during Ovulen therapy. Such fibroids may regress to pretreatment size after Ovulen is stopped. In the event of breakthrough bleeding the possibility of nonfunctional causes should be borne in mind. Additional means of contraception should be used during the hrst seven days of Ovulen administration in the first treated cycle, because early ovulation may possibly occur.

Side Actions—The following adverse reactions have been reported with Ovulen; however, a causal relationship to Ovulen administration has not been established in all of the listed complaints: headache, dizziness, depression, breast complaints, amenorrhea, chloasma, vomiting, allergy, edema, migraine, pulmonary embolism, thrombophlebitis, visual difficulties, nervousness, rash, itching, decrease in libido, tiredness, malaise, hair loss and hair growth. A small incidence of nausea, spotting and breakthrough bleeding has been reported; these complaints tend to diminish markedly or disappear after the first cycle of treatment. Some of these side actions have required discontinuance of the drug.

Dosage—One tablet daily for 20 consecutive days beginning 5 days after the onset of menstruation.

Before prescribing see Detailed Product Information, Ovulen.

An extensive list of references on Ovulen is included in the literature mailed to physicians.

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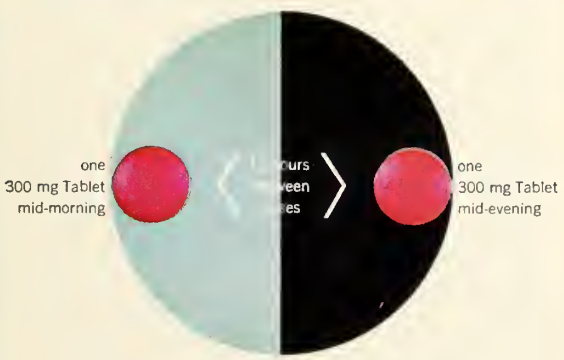


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Warning — In renal impairment, usual doses may lead to excessive systemic accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort.

Precautions and Side Effects — Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. Use of demethylchlortetracycline during tooth development (last trimester of pregnancy, neonatal period and early childhood) may cause discoloration of the teeth (yellow-grey-brownish). This effect occurs mostly during long-term use but has also been observed in short treatment courses. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment. Side reactions include glossitis, stomatitis, proctitis, nausea, diarrhea, vaginitis and dermatitis. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy. Anaphylactoid reactions have been reported.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

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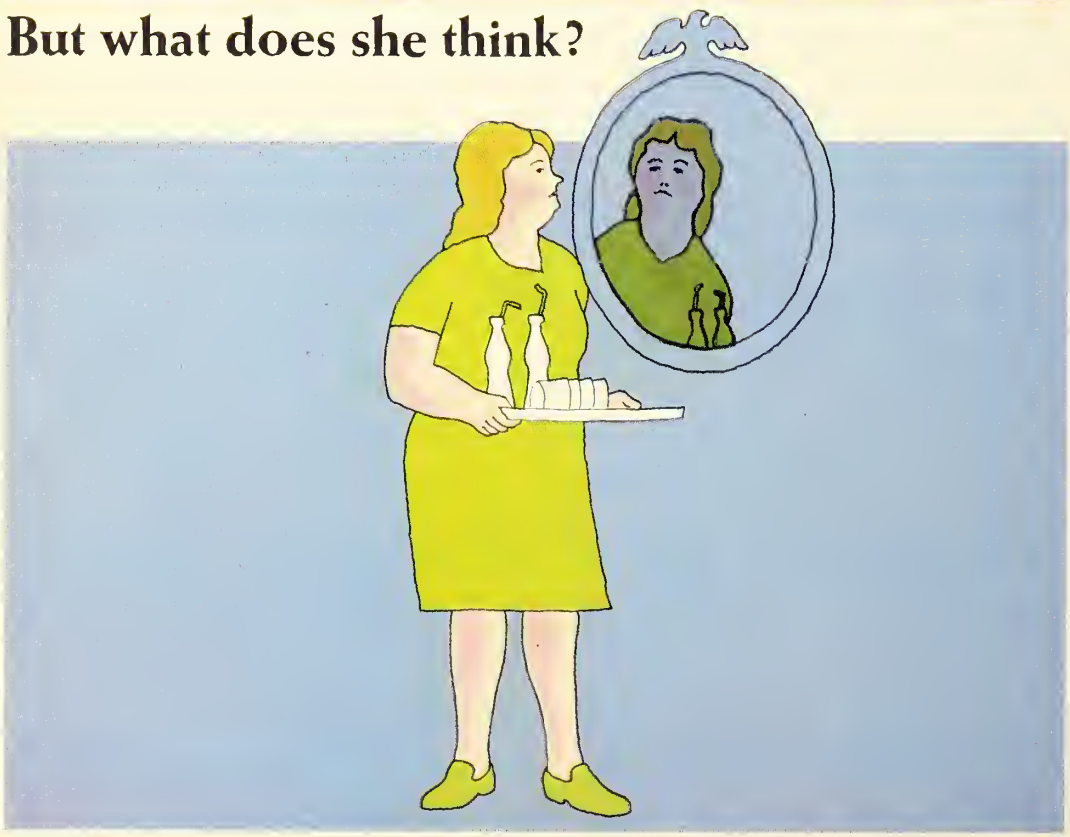
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Precautions: Use with caution in patients hypersensitive to sympathomimetic compounds, who have coronary or cardiovascular disease, or are severely hypertensive.

Dextro-amphetamine sulfate: Excessive use by unstable individuals may result in psychological dependence.

Meprobamate: Careful supervision of dose and amounts prescribed is advised, especially for patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, reduce dosage gradually. Sudden withdrawal may precipitate recurrence of preexisting symptoms such as anxiety, anorexia, or insomnia; or withdrawal reactions such as vomiting, ataxia, tremors, muscle twitching and, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, reduce dosage and avoid operation of motor vehicles, machinery or other activity requiring alertness. Effects of excessive alcohol consumption may be increased by meprobamate. Appropriate caution is recommended with patients prone to excessive drinking. In patients prone to both petit and grand mal epilepsy meprobamate may precipitate grand mal attacks. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side Effects: Overstimulation of the central nervous system, jitteriness and insomnia or drowsiness.

Dextro-amphetamine sulfate: Insomnia, excitability, and increased motor activity are common and ordinarily mild side effects. Confusion, anxiety, aggressiveness, increased libida, and hallucinations have also been observed, especially in mentally ill patients. Rebound fatigue and depression may follow central stimulation. Other effects may include dry mouth, anorexia, nausea, vomiting, diarrhea, and increased cardiovascular reactivity.

Meprobamate: Drowsiness may occur and can be associated with ataxia; the symptom can usually be controlled by decreasing the dose, or by concomitant administration of central stimulants. Allergic or idiosyncratic reactions: maculopapular rash, acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever, transient leukopenia. A case of fatal bullous dermatitis, following administration of meprobamate and prednisolone, has been reported. Hypersensitivity has produced fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, stomatitis, proctitis (1 case), anaphylaxis, agranulocytosis and thrombocytopenic purpura, and a fatal instance of aplastic anemia, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity, usually after excessive dosage. Impairment of visual accommodation. Massive overdosage may produce drowsiness lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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MOLECULAR REMODELING—

laboratory exercise or clinical necessity?

More than twenty-five years have passed since the discovery of the diuretic activity of sulfanilamide started pharmacologists on a succession of molecular remodelings to find the ideal diuretic.

Diuresis—a sought-after clinical effect from an unwanted side effect

It started in 1937 when a clinician reported that the administration of a sulfonamide was sometimes accompanied by an unexplainable side effect—metabolic acidosis.[1] Three years later the side effect was explained. The sulfonamide radical of sulfanilamide inhibited carbonic anhydrase,[2] the enzyme responsible for converting carbon dioxide and water to hydrogen ions and bicarbonate ions.

Later, other investigators showed by dog experiments that metabolic acidosis probably resulted when the inhibition of carbonic anhydrase upset the exchange of hydrogen and sodium ions, causing increased excretion of sodium as the bicarbonate.[3]

It was twelve long years after the first report of the unexplainable side effect (metabolic acidosis) that it was finally shown that large doses of sulfanilamide administered to edematous patients were indeed capable of promoting diuresis.[4] However, the possibility of toxic effects from its prolonged use and its relatively weak diuretic action made it impractical for clinical use as a diuretic.[5]

Because the inhibition of carbonic anhydrase seemed to be the key to effective diuresis, investigators began to look for more potent enzyme inhibitors —in the hopes that they would be more effective diuretics.

The most important of these early compounds, acetazolamide, enjoyed several years of fairly wide clinical use.

Its carbonic anhydrase inhibitory activity was several hundred times greater than that of sulfanilamide.[6] The increase in inhibitory activity, however, increased not only the excretion of sodium and bicarbonate ions, but also the excretion of potassium.[7] And, like its predecessor, acetazolamide precipitated mild acidosis. Its prolonged use could result in hypokalemic acidosis.[7]

The 'thiazides'—an answer to the metabolic acidosis caused by carbonic anhydrase inhibition

Despite the fact that the sulfonamide group appeared to be responsible for carbonic anhydrase inhibition which in turn appeared to be responsible for diuresis, investigators began to synthesize compounds with structural alterations to the sulfonamide group.

The first major breakthrough came with the synthesis of chlorothiazide. Altering the sulfonamide group did indeed alter the ability of chlorothiazide to inhibit carbonic anhydrase—it was only 1/10th as potent as acetazolamide in inhibiting the enzyme.[8] Despite the drop in inhibitory potency, however, chlorothiazide proved to be an effective diuretic—an observation that led to the conclusion that its diuretic action was due to some mechanism other than its action on carbonic anhydrase.[9,10]

For effective diuresis, chlorothiazide was administered in daily dosages ranging from 250 to 2000 mg.[11] It increased the excretion of sodium and chloride; and, to a lesser extent, potassium and bicarbonate.[11] The excretion of potassium appeared to be maximal at higher dose levels at which, theoretically, the carbonic anhydrase inhibitory effect is more active.[11] Its prolonged use, therefore, could sometimes result in metabolic hypokalemic, hypochloremic alkalosis.[7]

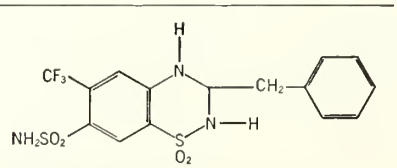
Naturetin—effective diuresis with more favorable electrolyte balance

Other thiazides followed—with improvements being aimed at two particular areas: 1. attempts to increase diuretic action in relation to the milligram potency of the drug, and 2. attempts at a more favorable sodium/potassium ratio in the urine, i.e., to decrease the excretion of potassium while maintaining the excretion of sodium.[12]

One of these, Naturetin, Squibb Bendroflumethiazide, has made advances on both these points. "By adding a 3-benzyl radical to hydroflumethiazide a rather dramatic reduction in dose range is accomplished. With this drug, effective sodium excretion is obtained with doses between 2.5 and 10 mg., which is a 200 to 1 ratio as compared to chlorothiazide..."[13]

Moreover, due probably to its virtual lack of carbonic anhydrase inhibition, Naturetin (bendroflumethiazide) has been shown to cause less potassium and bicarbonate loss and less alteration in urinary pH than either chlorothiazide or hydrochlorothiazide.

Naturetin is outstandingly effective not only in establishing, but also in maintaining, excretion of retained fluid in edematous patients. And its duration of action is sufficiently prolonged to allow a single daily administration in most patients. Naturetin is also an effective antihypertensive agent.



Contraindications: Severe renal impairment; previous hypersensitivity.

Warning: Ulcerative small bowel lesions have occurred with potassium-containing thiazide preparations or with enteric-coated potassium salts supplementally. Stop medication if abdominal pain, distension, nausea, vomiting, or G.I. bleeding occur.

Precautions: The dosage of ganglionic blocking agents, veratrum, or hydralazine when used concomitantly must be reduced by at least 50% to avoid orthostatic hypotension. Electrolyte disturbances are possible in cirrhotic or digitalized patients.

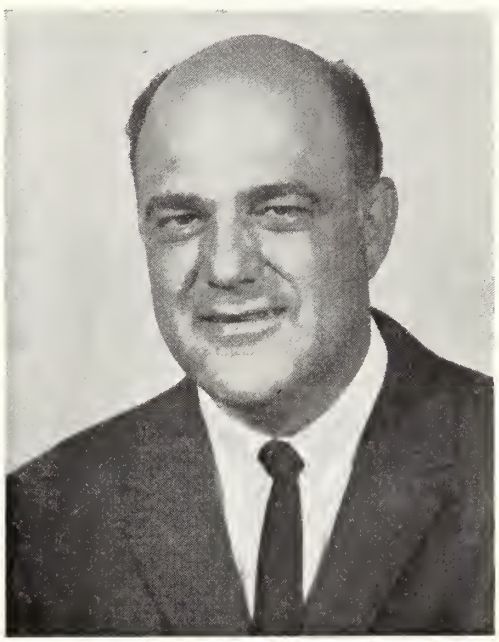
Side Effects: Bendroflumethiazide may cause increases in serum uric acid, unmask diabetes, increase glycemia and glycosuria in diabetic patients and may cause hypochloremic alkalosis, hypokalemia; cramps, pruritus, paresthesias, and rashes may occur.

Supplied: Naturetin (Squibb Bendroflumethiazide) 5 mg. and 2.5 mg. tablets. Also available Naturetin c K [Squibb Bendroflumethiazide (5 or 2.5 mg.) with Potassium Chloride (500 mg.)]. For full information, see Product Brief.

References: **1.** Southworth, H.: Proc. Soc. Exper. Biol. & Med. *36*:58, 1937. **2.** Mann, T. and Keilin, D.: Nature *146*:164, 1940. **3.** Pitts, R. F., and Alexander, R. S.: Am. J. Physiol. *144*:239, 1945. **4.** Schwartz, W. B.: New England J. Med. *240*:173, 1949. **5.** Friedberg, C. K., in Moyer, J. H., and Fuchs, M.: Edema Mechanisms and Management, Philadelphia, W. B. Saunders Co., 1960, p. 259. **6.** Cumming, J. R.; Tabachnick, E., and Seelig, M., in Moyer, J. H., and Fuchs, M.: op. cit., p. 254. **7.** Werko, L., in Moyer, J. H., and Fuchs, M.: op. cit., p. 188. **8.** Beyer, K. H., Jr., in Moyer, J. H., and Fuchs, M.: op. cit., p. 274. **9.** Maren, T. H., and Wiley, C. E.: J. Pharmacol. & Exper. Therap. *143*:230, 1964. **10.** Earley, L. E., and Orloff, J.: Ann. Rev. Med. *15*:149, 1964. **11.** Fuchs, M., and Mallin, S. R., in Moyer, J. H., and Fuchs, M.: op. cit., p. 276. **12.** Ford, R. V., in Moyer, J. H., and Fuchs, M.: op. cit., p. 290. **13.** cited in Fuchs, M., and Mallin, S. R. (ref. 11): op. cit., p. 283.

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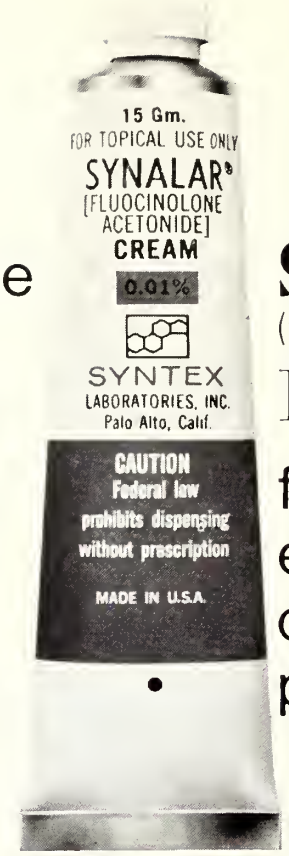
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Contraindications: Tuberculous, fungal, and most viral lesions of the skin (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of its components. **Precautions:** 1. *General*—Synalar Cream 0.01% is virtually nonsensitizing and nonirritating. Where severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. While topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use on pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time. 2. *Occlusive dressing method*—With occlusion of extensive areas, systemic absorption of the corticosteroid may occur, and suitable precautions should be taken. Occasional patients may show contact sensitivity to a particular dressing material or adhesive. Miliaria, folliculitis, or pyodermas have been seen infrequently with the use of this technique. The development of infection requires appropriate antibacterial therapy and discontinuation of the occlusive dressing method. Local atrophy and striae have been reported with protracted occlusive dressing therapy. While lesion relapses can be expected to occur in many psoriatic patients, remissions may persist for several weeks to several months in favorable cases. The patient whose psoriasis is in an active stage, with recent appearance of new lesions, may not be a good candidate and may show early relapse. Some plastic films may be flammable, and due care should be exercised in their use. Similarly, caution should be employed when such films are used on or left near children to avoid the possibility of accidental suffocation. **Side Effects:** Side effects are not ordinarily encountered with topically applied corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions. **References:** 1. Cahn, M. M., and Levy, E. J.: J New Drugs 1:262 (Nov.-Dec.) 1961. 2. Meenan, F. O.: J Irish Med Ass 52:75 (Mar.) 1963. 3. Robinson, H. M., Jr., Raskin, J., and Dunseath, W. J. R.: Southern Med J 56:797 (Jul.) 1963.

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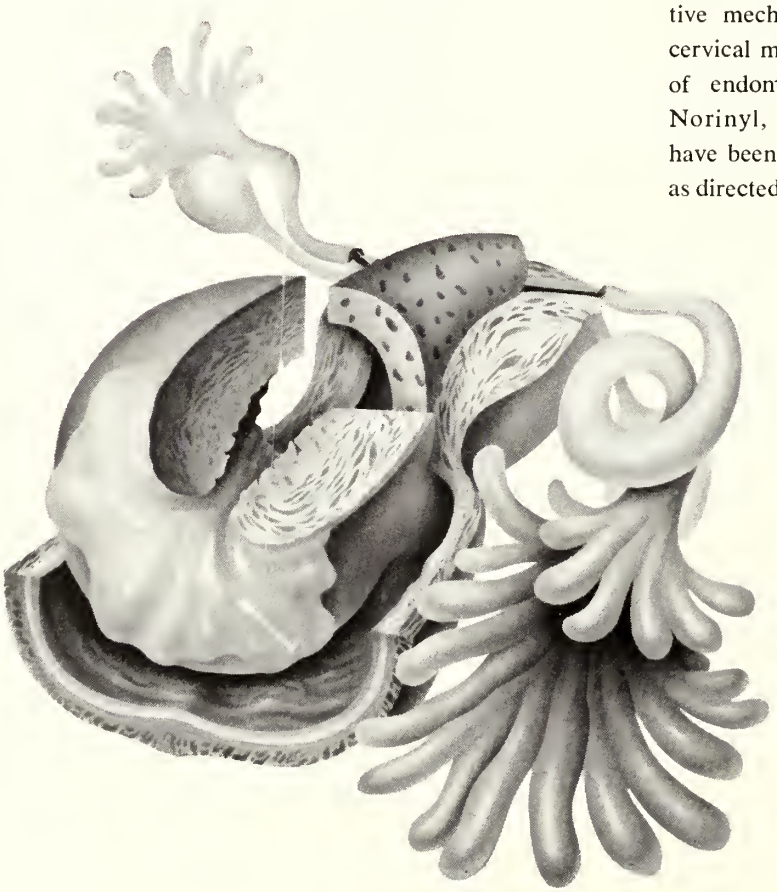
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inhibition of ovulation by means of 2 time-proved hormonal agents

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Contraindications: Thrombophlebitis or pulmonary embolism (current or past). Existing evidence does not support a causal relationship between use of Norinyl and development of thromboembolism. While a study which was conducted does not resolve definitively the possible etiologic relationship between progestational agents and intravascular clotting, it tends to confirm the findings of the Ad Hoc Advisory Committee appointed by the Food and Drug Administration to review this possibility. Cardiac, renal or hepatic dysfunction. Carcinoma of the breast or genital tract. Patients with a history of psychic depression should be carefully studied and the drug discontinued if depression recurs to marked degree. Patients with a history of cerebral vascular accident.

Warning: Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions, medication should be withdrawn.

Precautions: By May 1963, experience with norethindrone 2 mg.–mestranol 0.1 mg. had extended over 24 months. Through miscalculation, omission or error in taking the recommended dosage of Norinyl, pregnancy may result. If regular menses fail to appear and treatment schedule has not been adhered to, or if patient misses two menstrual periods, possibility of pregnancy should be resolved before resuming Norinyl. If pregnancy is established, Norinyl should be discontinued during period of gestation since virilization of the female fetus has been reported with oral use of progestational agents or estrogen. When lactation is desired, withhold Norinyl until nursing needs are established. Existing uterine fibroids may increase in size. In metabolic or endocrine disorders, careful clinical preevaluation is indicated. A few patients without evidence of hyperthyroidism had elevated serum protein-bound iodine levels, which in the light of present knowledge, does not necessarily imply hyperthyroidism. Protein-bound iodine increased following estrogen administration. Bromsulphalein retention has occurred in up to 25% of patients without evidence of hepatic dysfunction. Studies from 24-hour urine collections have shown an increase in aldosterone and 17-ketosteroids and decrease in 17-hydroxycorticoid levels. Thus, Norinyl should be discontinued prior to and during thyroid, liver or adrenal function tests. Because progestational agents may cause fluid retention, conditions such as epilepsy, migraine and asthma require careful observation. Thus far no deleterious effect on pituitary, ovarian or adrenal function has been noted; however, long-range possible effect on these and other organs must await more prolonged observation. Norinyl should be used with caution in patients with bone, renal or any disease involving calcium or phosphorus metabolism.

Side Effects: Intermenstrual bleeding; amenorrhea; symptoms resembling early pregnancy, such as nausea, breast engorgement or enlargement, chloasma and minor degree of fluid retention (if these should occur and patient has not strictly adhered to medication plan, she should be tested for pregnancy); weight gain; subjective complaints such as headache, dizziness, nervousness, irritability; in a few patients libido was increased. In a total of 3,090 patients, 2.2% discontinued medication because of nausea.

NOTE: See sections on contraindications and precautions for possible side effects on other organ systems.

Dosage and Administration: One Norinyl tablet orally for 20 days, commencing on day 5 through and including day 24 of the menstrual cycle. (Day 1 is the first day of menstrual bleeding.)

Availability: Dispensers of 20 and 60 tablets; bottles of 100.

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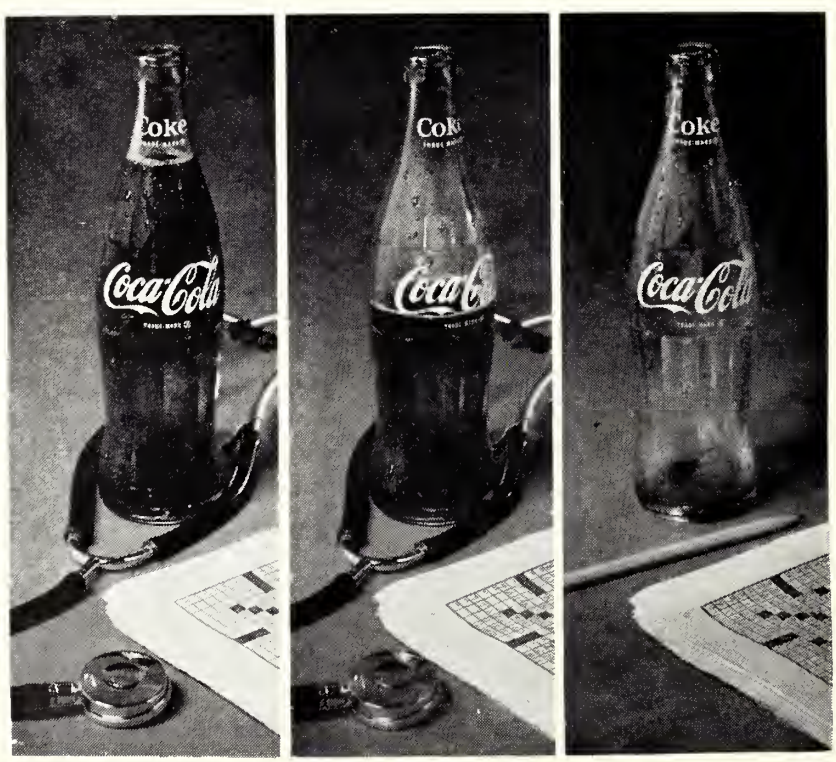
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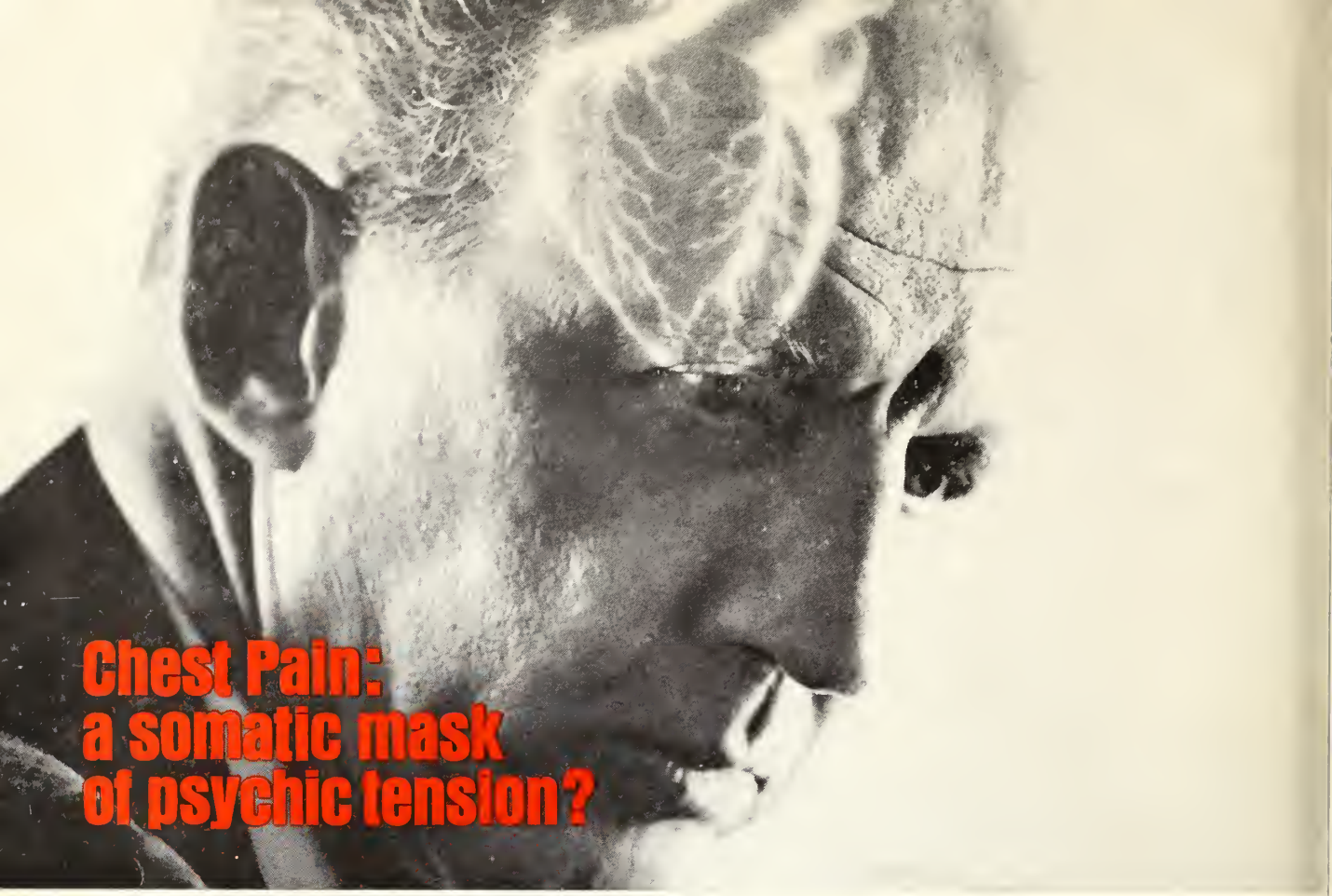
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Valium (diazepam) is generally well tolerated, and usually does not impair mental acuity or ability to function. If side effects such as ataxia and drowsiness occur, they usually disappear with dosage adjustment.

Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount in elderly patients (not more than 1 mg, one or two times daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established, driving during therapy not recommended. In general, concurrent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepatic function and in patients who may be suicidal, periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash, paradoxical reactions (excitement, depression, stimulation, sleep disturbances and hallucinations) and changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage — Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hrs, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

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